A dissertation on

TOWARDS DEVELOPING A SCORING SYSTEM FOR RISK STRATIFICATION OF YELLOW COWDUNG COLORING AGENT POISONING AND TO ASSESS THE NEED FOR INTENSIVE CARE TREATMENT

Dissertation submitted to

THE TAMIL NADU Dr M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMIL NADU

With partial fulfillment of the regulations required

For the award of degree of

M.D. GENERAL MEDICINE

BRANCH- I

COIMBATORE MEDICAL COLLEGE,

COIMBATORE

May 2018

CERTIFICATE

This is to certify that this dissertation titled **"TOWARDS DEVELOPING A SCORING SYSTEM FOR RISK STRATIFICATION OF YELLOW COWDUNG COLORING AGENT POISONING AND TO ASSESS THE NEED FOR INTENSIVE CARE TREATMENT"** has been done by **Dr. SHADIYA C** under my guidance.

Further certified that this work is an original study of bonafide cases.

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The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation Proposal is accepted / ~~Not~~ accepted and you are permitted / ~~Not~~ ~~permitted~~ to proceed with the above Study.

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22.4.16

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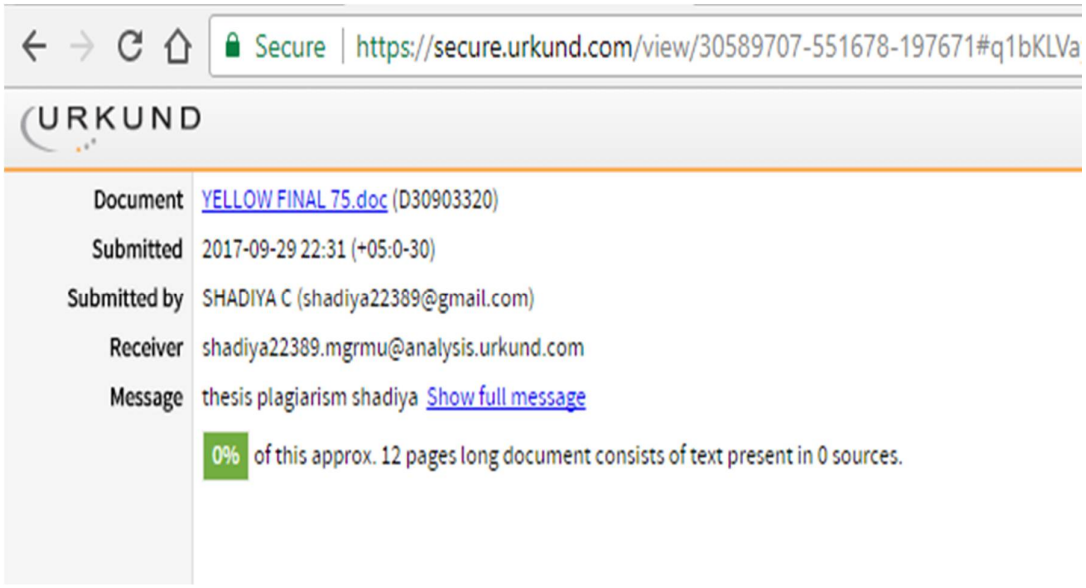

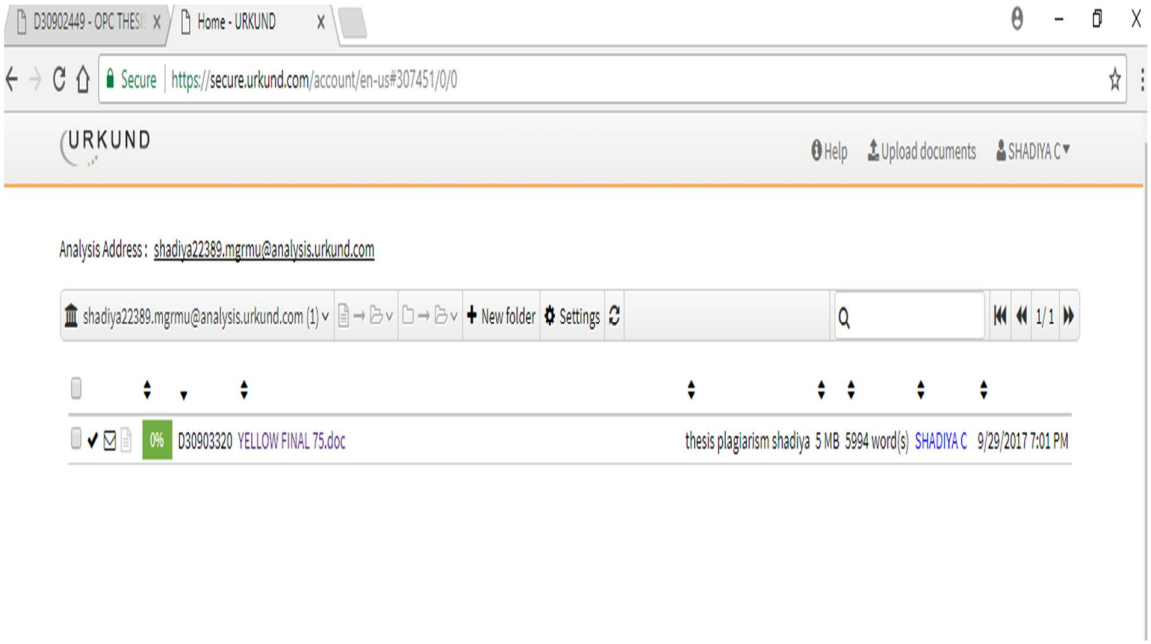

DECLARATION

I, Dr. SHADIYA C, declare that the Dissertation titled " **TOWARDS DEVELOPING A SCORING SYSTEM FOR RISK STRATIFICATION OF YELLOW COWDUNG COLORING AGENT POISONING AND TO ASSESS THE NEED FOR INTENSIVE CARE TREATMENT**" Submitted to the Dr.MGR Medical university, Guindy, Chennai is an original work done by me during the academic period from December 2015- July – 2017 at the Department of Medicine, Coimbatore Medical College Hospital, Coimbatore, under the guidance and direct supervision of Dr. USHA.S in partial fulfillment of the rules & regulations of the Dr.MGR Medical University for MD Medicine post graduate degree.

All the details of the patients, the materials and methods used are true to the best of my knowledge.

I assure that this dissertation has not been submitted to or evaluated by any other Medical University.

Dr. SHADIYA.C.

ACKNOWLEDGEMENT

I wish to express my sincere thanks to our respected **Dean Dr. B.ASOKAN, M.S, McH** for having allowed me to conduct this study in our hospital .I express my heartfelt thanks and deep gratitude to the Head of department of medicine Prof **Dr KUMAR NATARAJAN, MD** for his generous help and guidance in the course of the study.

I'm thankful to my unit chief **Dr. S.USHA, M.D** for her valuable help and encouragement for doing my study. I sincerely thank all **PROFESSORS, ASSISTANT PROFESSORS Dr. AVUDAIAPPAN.S, Dr. BALAMURUGAN.P, Dr. ALAGU THIYAGARAJAN** for their guidance and kind help.

My sincere thanks to all my friends and post graduate colleagues for their whole hearted support and companionship during my studies.

I thank my patients who formed the back bone of this study without whom this study would have not been possible.

Last but not the least I thank my parents for having extended unconditional support throughout my life.

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ABBREVIATION

WHO	–	World Health Organization
RBC	–	Red Blood Cell
CVS	–	Cardiovascular System
GIT	–	Gastro Intestinal Tract
CNS	–	Central Nervous System
TAC	–	Total Antioxidant Capacity
RBS	–	Random Blood Sugar
ACTH	–	Adreno Cortico Tropic Hormone
LDL	-	Low Density Lipoprotein
VLDL	-	Very Low Density Lipoprotein
HDL	-	High Density Lipoprotein
ECG	–	Electro Cardio Graph
LDH	-	Lactate Dehydrogenase
CPK	–	Creatine Phosphokinase
APACHE II	-	Acute physiology and chronic health evaluation II
SGOT	–	Serum Glutamic Oxaloacetic Transaminase
SGPT	–	Serum Glutamic Pyruvic Transaminase
ICU	–	Intensive Care Unit
NAC	–	N Acetyl cysteine
GCS	-	Glasgow coma scale

INTRODUCTION

Yellow cow dung is a dye, chemically auramine O is a highly lethal poisoning with no known available anti dote. Chemically it is auramine o, a highly lipophilic chemical which produces neurotoxicity, cardiotoxicity, hepatotoxicity and nephrotoxicity when consumed in lethal amount. From time immemorial people from rural areas tend to use cowdung for cleaning of their premises because of its germicidal and antiseptic properties. In recent times due to unavailability of natural cow dung people started using its synthetic variety.

There are two synthetic varieties of cow dung available which are (1) yellow cow dung –auramine O and (2) green cow dung –malachite green.

Even though the manufacturing and selling of these synthetic dyes are banned by law long back, it is still available in the rural areas of southern Tamilnadu. More and more cases are getting reported with synthetic dye poisoning. when consumed in large quantities these are lethal .As this poisoning is largely confined to certain pockets of the state ,not much studies or researches has been done in this regard. In this study we mainly concentrated on the yellow cow dung (auramine O) poisoning.

It is highly cardiotoxic causing various cardiac manifestations. out of which arrhythmias especially AV arrhythmias and AV conduction blocks are life threatening. It can also cause hepatitis, centilobular necrosis and acute liver cell failure. Renal failure is also occur in yellow poisoning but it is much more common in green cow dung poisoning because of its hydrophilic properties.

FIG 1 – COW DUNG USE IN RURAL AREAS

Even though the poisoning is lethal, no prognostic guidelines or treatment protocols govern the management of yellow cowdung poisong .Not much studies are available describing the clinical manifestations, symptoms and signs. No risk stratification protocols are available in order to classify as mild, moderate and severe poisoning.

Treatment with N-Acetyl cysteine is giving some promising results because of its anti oxidant properties. In this following study we tried to elucidate the warning signs and symptoms of yellow cow dung poisoning and to formulate a scoring system for risk stratification.

AIMS AND OBJECTIVES

AIM

The emergency medicine department of Coimbatore Medical College receives 2-3 cases of yellow cow dung coloring agent poisoning on a daily basis. The purpose of this study is to develop an improved scoring system for the risk stratification of yellow cow dung coloring agent poisoning and to assess the need for intensive care treatment.

OBJECTIVES

- To study the clinical and biochemical profile of yellow colouring agent poisoning.
- To develop a scoring system for risk stratification of yellow cow dung poison and to assess the need for intensive care.

REVIEW OF LITERATURE

Yellow coloring powder is a dye used in chemical industry, is a highly toxic poison with no known definite anti dote available. Traditionally the cow dung is derived from the excreta of cows. It was used in the rural areas for cleaning the household premises and also the temple premises. It has many useful properties. Cow is worshipped as a holy animal in many parts of India. It has a manure property used in agriculture. According to Ayurveda and vedic literature, Gomaya/ cowdung is used as a biomedical manure. It has great organic property.

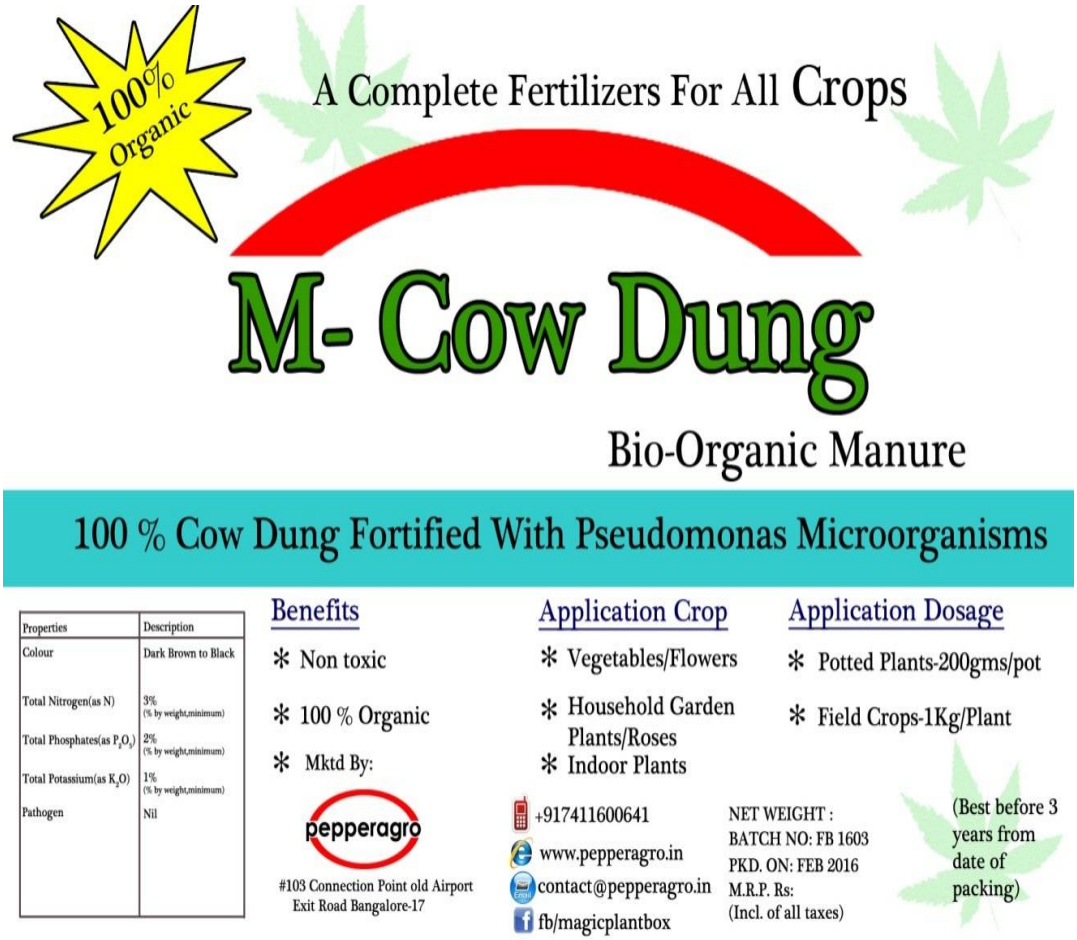
FIG 2 – COW DUNG USED AS BIO FUEL

These days the organic pollutants and the synthetic pesticides make a great harm to the aquatic land and the atmosphere. They are also harmful to the ecosystem of the earth. They pollute the human soil, and water and make the environment a bad place to live. Statistics suggest that around 30 metric tons of pesticidal waste is disposed in the soil and the water. Pesticide residues in the soil are above the permissible levels. The incidence of various diseases including carcinomas has been on a rise in the latter half of 20 th century. The persistence of synthetic zenobiotics in the environment causes dramatic public, ecologic and pharmaceutical regulatory concern causing increase in the number of potential toxins, mutagens and carcinogens. This will affect the environment in a drastically negative way.

FIG 3 – COW DUNG AS FERTILIZER

Yellow cow dung which is derived from the cows and bullocks is dried powdered and packeted and is now available commercially also for use as fertilizer and a naturally available biomass fuel in many parts of the rural India, it is the main source of fuel to light the houses, run the motor, draw water from the well and other house hold and domestic purposes.

FIG 4 – COW DUNG AS BATH SOAP AND SHAMPOO



Cow dung is also now marketed in the form of soap, detergent and shampoo. The natural cow dung is processed and is fortified with pseudomonas and other microorganisms to make it as a synthetic as well as natural product.

Cow dung is also used to treat skin diseases, wounds and cuts in certain parts of rural India. However its use as an antiseptic or disinfectant is still under a debate as it is often contaminated with the pathogens in the soil. There are case reports of even tetanus being in patients doing these practices.

These days because of the rarity of cows in the urban areas the traditional cow dung was replaced by synthetic preparations. These synthetic preparations are produced in the dyeing industries and other small scale industries for commercial preparation. These days these synthetic preparations are freely available in all shops in the rural India as well as in cities. It is well known in Tamilnadu in the local language as SAANI POWDER.

The sale of this synthetic coloring agent has been banned legally in India. but no strict laws govern its implementation. It is freely available in homes and it is sometimes consumed by children accidently. In Tamilnadu the poisoning of this synthetic compound id most common in Coimbatore, Avinashi and Erode. Many deaths has been reported in these area the incidence is on an increase in other parts of Tamilnadu and certain parts of rural north India, the easy availability of the compound across all the shops and the cheaper cost make this poisoning as one of the most common .

Chemically it is available in two different colors

(1) Auramine_0

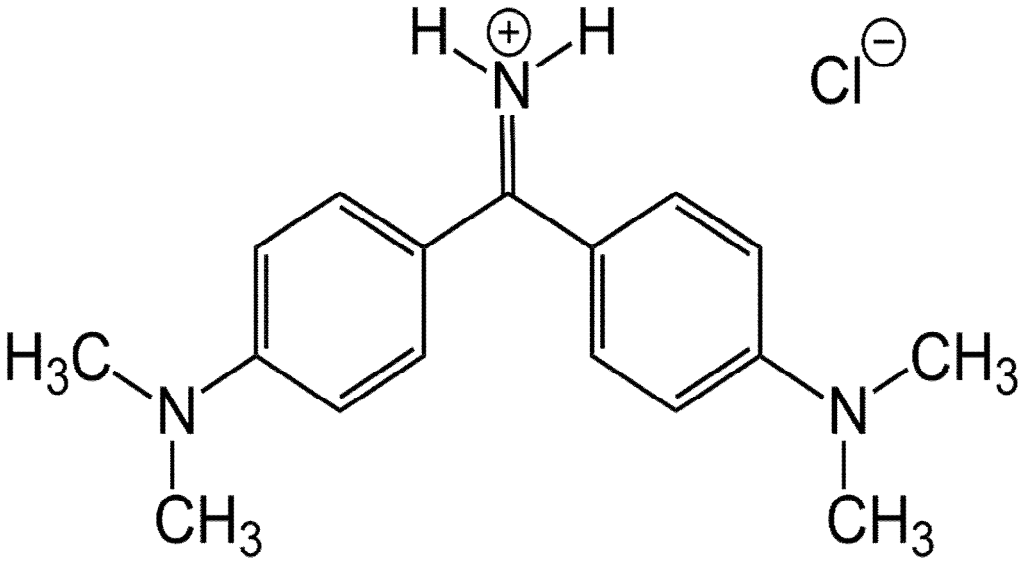
(2) Malachite green

Auramine _0 is used as a coloring compound in dyeing industry in this part of Tamilnadu.

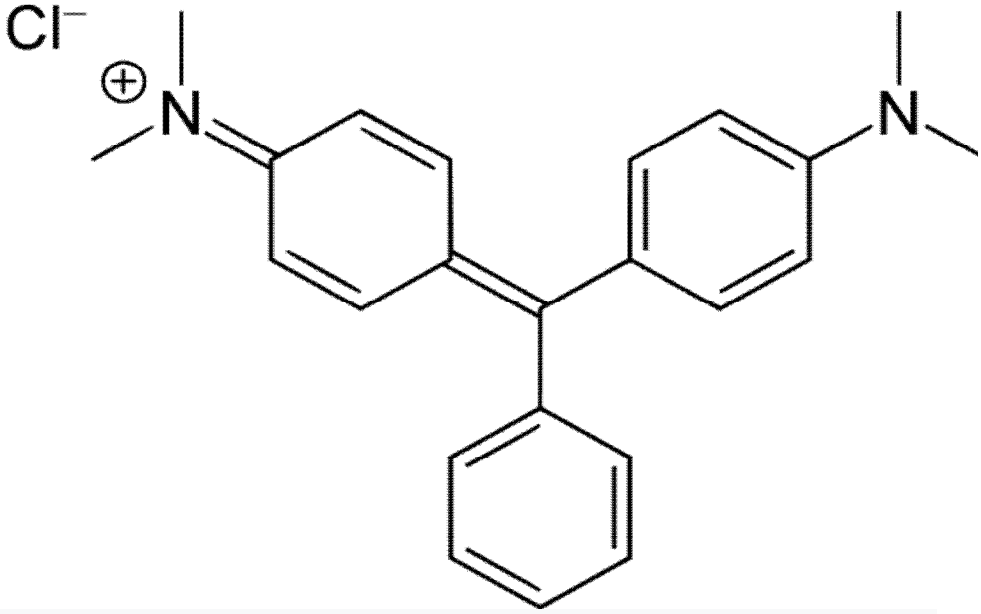
Auramine 0 is water soluble and lipophilic. It is used in microbiology to stain AFB (mycobacterium tuberculosis, mycobacterium avium, mycobacterium kansesi, Imycobacterium, scrofulaceum, and mycobacterium gordonei). It has strong affinity to the cell wall mycolic acid components .it is also used as an additional stain for the Schiff reagent.

It has amino methyl group that will undergo conjugation to positively charged amino atom and it leads to hydroxylation. The direct toxicity of Auramine_0 is to induce deoxyribonucleic acid damage in the liver, kidney, and the stem cells. The carcinogenicity of the compound has been proven in many studies.

Malachite green is used in the fishing industry as a synthetic parasiticide. To the surprise there are no warning labels on the compounds which are sold. only very few case reports has been documented. There is no definite anti-dote for it. The mechanism of action of the toxin, the clinical features of the poison, the complications of the poison and the pathophysiology of the toxicity is not clearly understood.



(bis[4-(dimethylamino) phenyl] methaniminium chloride)



(4-{[4-(Dimethylamino)phenyl](phenyl)methylidene}-*N*,*N*-dimethylcyclohexa-2,5-dien-1-iminium chloride)

Chemical formula of yellow and green coloring agent powder is shown.

FIG 5 – COLORING AGENT AVAILABLE IN PACKET IN SHOPS (YELLOW & GREEN)

TOXICITY PROFILE

Auramine is highly lipophilic and can cross the BBB and can cause central nervous system effects like depressed consciousness, increased intracranial tension and seizures. It causes metabolic abnormalities and increased blood glucose levels. It can cause depressed cardiac function, hypotension and arrhythmias. Sinus arrhythmias are seen in majority of cases and in severe forms of toxicity are seen cases of severe poisoning. It is hepatotoxic as evidenced by presence of nausea, vomiting and increases serum transaminase levels. Serum bilirubin levels were also increased in cases with severe toxicity. In severe cases, it also caused elevated serum creatinine and blood urea. The mechanism of acute kidney injury is by direct cardiac depression effect causing hypotension, reduced contractility, causing negative dromotropic action, causing rhythm disturbances and by metabolic acidosis.

Because of its local irritant property, it can cause irritation of the mucosa of the skin, eyes. It can cause corneal and conjunctival abrasions and in severe cases can lead to blindness. Absorption can also occur through skin because of the consumption of the poison when it is mixed with the hand.

It is cheap and is available across all the shops and costs only R.s 5 per packet. The lethal dose of the poison is 0.5 mg per kg body weight.

The chemical compound has no definitive proportions and is usually manufactured in small scale industries. The composition of the compound varies from place to place and from manufacturer to manufacturer. This allows for the present day need for the urgent research to analyze the chemical structure of the compound and strict measures to govern the production restriction.

The chronic exposure of this toxin in persons working in the manufacturing plants leads to various mutagenicity and high incidence of carcinomas especially cancers of the bladder, lymphoproliferative system malignancy and other hematologic malignancy. Case reports also shows the presence of intrauterine growth abnormalities in the fetus or persons exposed chronically.

APACHE II

Apache II scoring system is used to classify patients with many systemic diseases. The classification is useful in patients admitted in intensive care units and also poisoning. According to APACHE II scoring we can classify the severity of poisoning as mild, moderate and severe. The prognosis of the patients largely depends upon the APACHE II severity. As we know this scoring system includes 12 parameters including chronic medical condition and age. In our study we used APACHE II scoring system for ascertaining the severity of poisoning.

But from the table given below it is very well understood that as this criteria involve a large number of parameters including arterial blood gas analysis, blood pressure, heart rate, respiratory rate, sodium, potassium, creatinine, hematocrite, white blood count and consciousness .Each parameter is again divided into positive and negative extremes. And each of them was given a point. After calculating points for each variable, we will get a total value. Depending upon this value we can categorize the patient from mild to severe groups.

As calculation of severity by using APACHE II is rather a hectic process. This cannot be implemented in emergency settings. These lacunae made us to find out a whole new scoring system solely for yellow cow dung coloring agent poisoning.

The APACHE II Severity of Disease Classification System

Physiologic Variable	+4	+3	+2	+1	0	+1	+2	+3	+4
Temperature - rectal (°C)	≥41	39-40.9		38.5-38.9	36-38.4	34-35.9	32-33.9	30-31.9	≤29.9
Mean Arterial Pressure (mm Hg)	≥160	130-159	110-129		70-109		50-69		≤49
Heart Rate	≥180	140-179	110-139		70-109		55-69	40-54	≤39
Respiratory Rate (nonventilated or ventilated)	≥50	35-49		25-34	12-24	10-11	6-9		≤5
Oxygenation (mmHg) a. $FiO_2 > 0,5$ use A-aDO_2 b. $FiO_2 < 0,5$ use PaO_2	a ≥500	350-499	200-349		<200				
	b				> 70	61-70		55-60	<55
Arterial pH	≥7.7	7.6-7.69		7.5-7.59	7.33-7.49		7.25-7.32	7.15-7.24	<7.15
Serum Sodium (mmol/l)	≥180	160-179	155-159	150-154	130-149		120-129	111-119	≤110
Serum Potassium (mmol/l)	≥7	6-6.9		5.5-5.9	3.5-5.4	3-3.4	2.5-2.9		<2.5
Serum Creatinine (mg/dl, Double point score for acute renal failure)	≥3.5	2-3.4	1.5-1.9		0.6-1.4		<0.6		
Hematocrit (%)	≥60		50-59.9	46-49.9	30-45.9		20-29.9		<20
White Blood Count (in 1000/mm³)	≥40		20-39.9	15-19.9	3-14.9		1-2.9		<1
Glasgow-Coma-Scale (GCS)	Score = 15 minus actual GCS								
Serum HCO_3 (venous, mmol/l, use if no ABGs)	≥52	41-51.9		32-40.9	22-31.9		18-21.9	15-17.9	<15
A = Total Acute Physiology Score APS	Sum of the 12 individual variable points								

B = Age Points		**C = Chronic Health Points**
≤44 years	0 points	If the patient has a history of severe organ system insufficiency or is immunocompromised assign points as follows:
45-54 years	2 points	a. For nonoperative or emergency postoperative patients – 5 points
55-64 years	3 points	b. For elective postoperative patients – 2 points
65-74 years	5 points	
≥75 years	6 points	

APACHE II Score = Sum of A (APS points) + B (Age points) + C (Chronic Health points)

(From: Knaus WA, Draper EA, Wagner DP, Zimmerman JE. APACHE II: a severity of disease classification system. Crit Care Med 1985;13(10):818-29)

The APACHE II Score

Chronic Health Points

History of severe organ insufficiency	Points
Non-operative patients	5
Emergency postoperative patients	5
Elective postoperative patients	2

- Organ insufficiency or immunocompromised state must have preceded the current admission
- Immunocompromised if:
  - Receiving therapy reducing host defences (immunosuppression, chemotherapy, radiation therapy, long term steroid use, high dose steroid therapy) or
  - Has a disease interfering with immune function such as malignant lymphoma or leukaemia
- Hepatic insufficiency if:
  - Biopsy proven cirrhosis
  - Portal hypertension
  - Episodes of upper GI bleeding due to portal hypertension
  - Prior episodes of hepatic failure, coma or encephalopathy
- Cardiovascular insufficiency if:
  - New York Heart Association Class IV
- Respiratory insufficiency if:
  - Severe exercise restriction due to chronic restrictive, obstructive or vascular disease,
  - Documented chronic hypoxia, hypercapnia, secondary polycythaemia, severe pulmonary hypertension
  - Respirator dependency
- Renal insufficiency if:
  - On chronic dialysis

CLINICAL SIGNS AND SYMPTOMS

People take yellow cow dung coloring agents mainly for suicidal purposes,a few accidental ingestions were also repoted . The use of yellow cow dung for homicidal purpose is not common. They will consume the poison mixed with water or many of the time with alcohol. Double poisoning as evidenced by mixing it with other poisons like organophorphoros componds are also common.

SYMPTOMS	SIGNS
NAUSEA	TACHYCARDIA
VOMITING	TACHYPNOEA REQUIRING VENTILATOR SUPPORT
ABDOMINAL PAIN	ICTERUS
LOOSE STOOLS	OLIGURIA
IRRITABILITY	HYPOTENSION.
ALTERED SENSORIUM	ARRYTHMIAS
SEIZURES	LOW GCS
EPIGASTRIC PAIN	TEMPERATURE
CONFUSION	EPIGATRIC TENDERNESS
HIGH COLOURED URINE	BASAL CREPTS
TENESMUS	
PURPURIC SPOTS	
MALENA	

Majority of patients presents with yellowish discolouration of skin, as a part of poisoning. They will have symptoms of nausea, vomiting, abdominal pain and loose stools.These symptoms are present uniformely in every patients irrespective of their severity of poisoning.moderaate to severe cases will present with hypotension,tachycardia,reduced consciouss level,and arrhythmias.

FIG 6 – STAINING OF FACE AND BODY IN POISONING CASE

They can have gastrointestinal symptoms as a side effect of local irritant action of yellow cowdung poisoning. After admission if the symptoms are persisting there is high chance of development of toxin

induced liver injury mainly due to centrilobular necrosis of hepatocytes.they can have elevated bilirubin values,elevated liver enzymes(SGOT,SGPT) and elevated alkaline phosphatase values.

As becase of lipid solublitity of the poison it can produce early central nervous system effects pruducing seizures.They will have generalised tonic clonic seizures and can go for status epilepticus. Once they develop status epilepticus prognosis will be poor.They will present with low Glas gow coma scale. Patients can also present with metabolic abnormalities and some of them may need intubation.

Yellow cowdung is a well known cardiotoxic dye.which can cause tachycardia, hypotension and cardiac arrythmias. Sinus arrythmias will be present in mild ,moderate and severe poisoning. In severe poisoning they can have atrioventricular arrythmias,left bundle branch and right bundle branch block pattern sometimes they can go for complete heart blocks.

They can go for nephrotoxicity by direct action on toxins on renal tubules and also due to associated hypotension occuring as a result ofrepeated vomiting. They can have elevated blood urea and serum creatinine values.some of them may need immediate dialysis because of renal failure.

FIG -7 YELLOWISH RYLES TUBE ASPIRATE

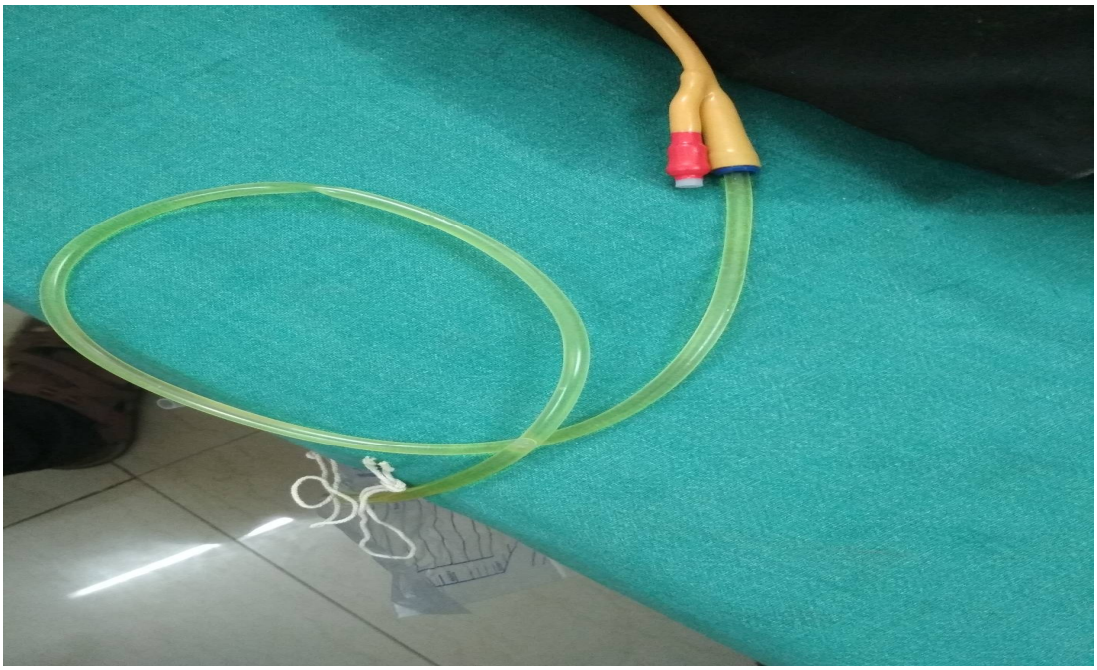


FIG 7 - FOLEYS CATHETER SHOWING YELLOW COLOR URINE

TREATMENT OF YELLOW COLORING AGENT POISONING

The principle idea of poison management is that all poisoning patients should be managed in such a way that they have a serious intoxication even though clinical manifestations are minimal.

Steps of management of acute poisoning.

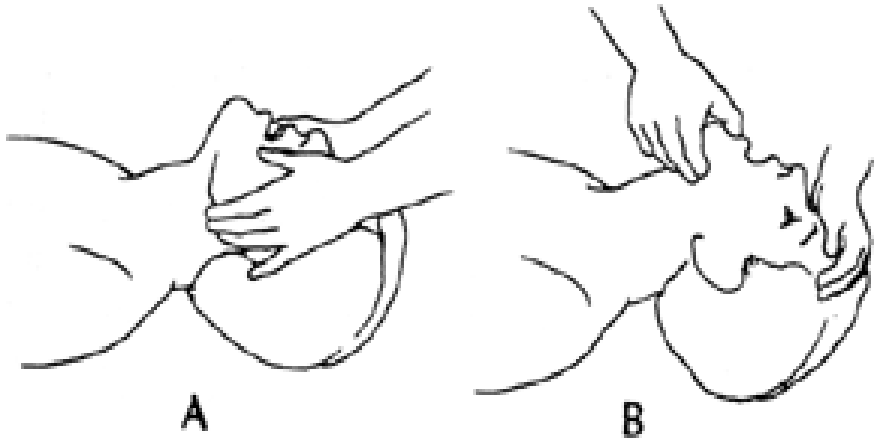
I. ROUTINE CARE:

The routine poison management comprise of maintaining airway, breathing and circulation.The initial ABCDE method should be applied to all poisoning patients. Many of times poison patients only require adequate supportive care to tide over the critical situations.

- **AIRWAY**

Most common factor leading to death in manhy poisoning is an obstructed airway.the mojor contributing factor for an obdstruycted airway is loss of tone of tongue,aspiration of gastric contents,respiratory muscle paralysis may be due to effect of poisoning or as a complcation of aspiration pneumonitis.

For every patient we have to make sure that there is no falling backward of tongue obstructing the airway.the neck lift and jaw thrust manuvre should be applied for patients with suspected obstruction.



INDICATIONS OF ENDOTRACHEAL INTUBATION

In a pateint with poisoning includes

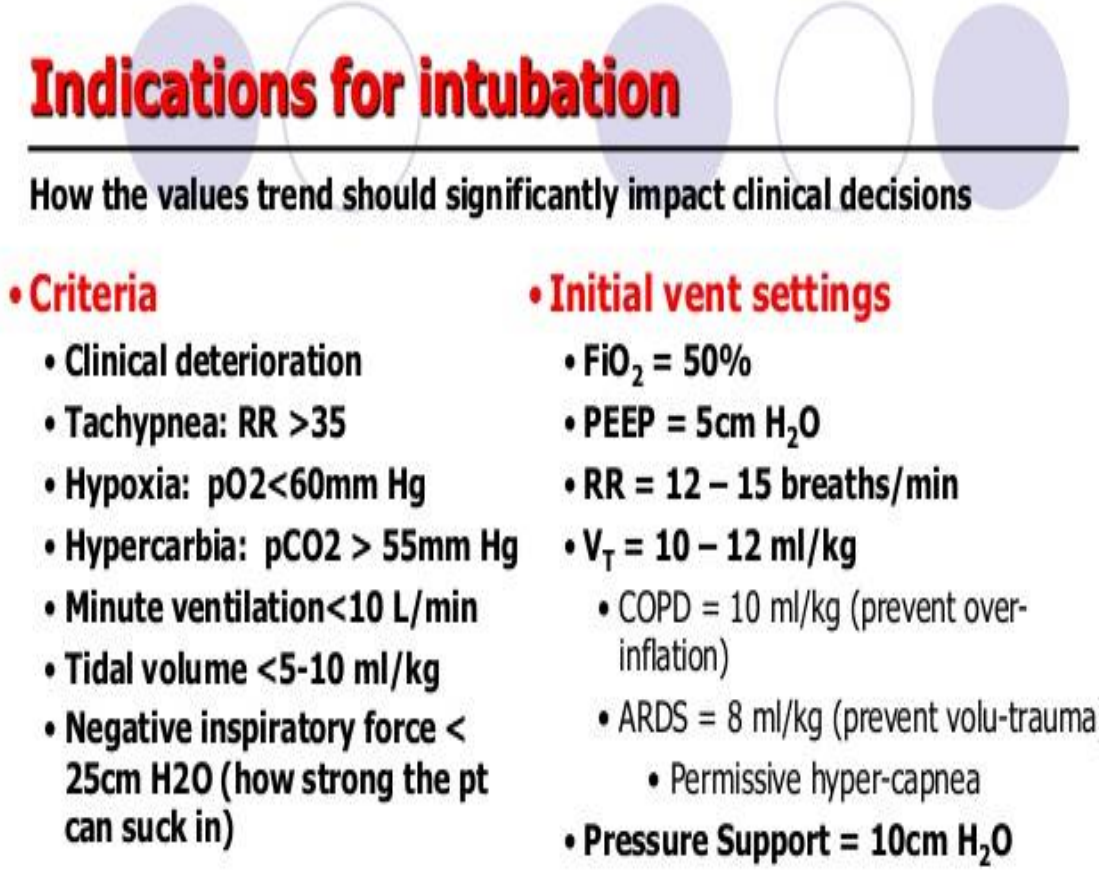
- Low GCS score,absent gag reflexes,to prevent the possibility of aspiration while giving a stomach wash.
- In patients with respiratory muscle paralysis,respiratory failure.
- For the purpose of removal of secretions in patients with toxin induced pilmonary edema.
- To administer PEEP therapy for those patients with suspected ARDS.
- To prevent falling back og tongue by securing a oropharyngeal airway in comatose patients.

Standard Criteria for Initiating Mechanical Ventilation

- Apnea or absence of breathing
- Acute respiratory failure
- Impending respiratory failure
- Refractory hypoxic respiratory failure
- Ventilatory insufficiency and the need to protect the airway or manage secretions

Table I – Weaning protocol

Protocol	Time of nebulization Period		
	Morning	Afternoon	Night
Respirator F = 2 rpm PSV = 10cmH_2O FIO2 = 0.4 PEEP= 5cmH_2O	30min 1 h 2 h 3 h 4 h 5 h 6 h	30min 1 h 2 h 3 h 4 h 5 h 6 h	Respirator Respirator 1 h 2 h 3 h 4 h 5 h
	24 h		



(2)BREATHING:

Monitoring the respiratory rate is important Patients can have tachypnoea,hypoxia,hypercarbia,bronchospasm and respiratory failure.

Normal respiratory rates:

AGE	BREATHS/MIN
NEWBORN	30-60
INFANT	25-40
TODDLER	20-30
YOUNG CHILDREN	20-25
OLDER CHILDREN	15-20
ADULTS	12-20

(3)CIRCULATION:

Always check pulse and blood pressure,secure an IV line and start the patient on IV fluis ,normal saline or any isotonic crystalloiuds solution is preferred. Patient should be connected to a continuous ECG monitoring in order to identify development of arrythmias.

(4)DISABILITY:

Depressed level of sensorium is a common feature of posoning.

Patients with altered sensorium or respiratory depression of unknown etiology will benefit from administration of COMA COCKTAIL.

Which is a mixture of

1.100% xygen.

2.Naloxone.

3.Glucose.

4.Thiamine.

(5) EXPOSURE:

Further absorption of poison should be prevented.Dermal exposure prevented byremoval of all the clothes soiled with poison,washing gently with soap and water atleast for 30 minutes.Washing conjunctiva with water for 20 minutes will help removing conjuctival injury. Gastrointestinal decantamination done by induction of emesis,gastric lavage and activated chacoal.

1.INDUCTION OF EMESIS.

Syrup ipecac is the safe method for inducing emesis in poisoning. Dose :30 ml should be given initially.If emesis does not occur within 30 minutes of administration ,move around the patient,if still no emesis occuring then a second dose can be given.Emesis is contraindicated in convulsions,corrosives,sharp objects,comma, reduced reflexes, recent surgical interventions, hemorrhagic tendencies, less than 6 months of age.

2.GASTRIC LAVAGE:

It is very effective when administered within 6 hours of ingestion.it can be used when there is contraindications for emesis or when emesis is failed.

It is contraindicated in posoning of

- Corrosives
- Coma
- Convulsions
- Petroleum distillates
- Oesophageal varices

FIG 8 – GASTRIC LAVAGE TUBE

3.ACTIVATED CHARCOAL:

It has got high adsorbing capacity. It prevents further absortion of poison from the gastrointestinal tract and reducing further toxicity.

Dose:1 mg/kg

Multidose activated charcoal is the more effective way of administering charcoal.

Administering more than 2 doses of chacoal for the treatmengt of given posoning

- Contraindications
- Coma
- Intestinal obstruction
- Corrosives
- If an oral antidote is given.
- Hydrocarbons

4. CATHARTICS(LAXATIVES):

These are substances which enhances the GI motility hence decreases the time for GI absorption.Two types are available osmotic and irritant cathartics.

Contraindications:

- GIT hemorrhage
- Recent bowel surgery

- Intestinal obstruction
- Renal failure

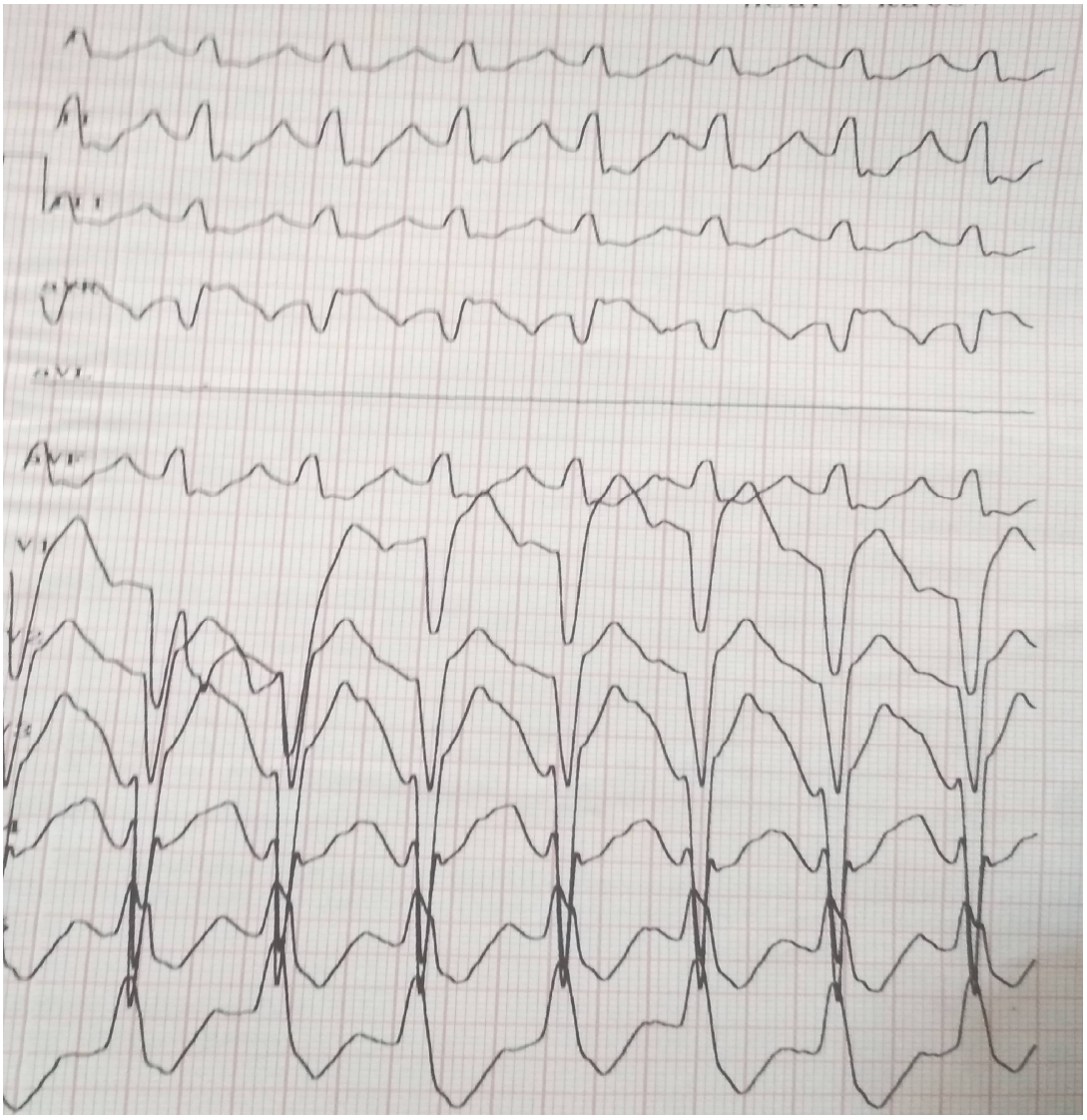
II . SUPPORTIVE TREATMENT

1.HYPOTENSION

Intravenous crystalloids should be administered at the rate of 20ml/kg and should be continued till the signs of hypotension recovers and patient has got adequate urine out put.In case of persistent hypotension even after fluid management , patient should be put on adequate inotropic support, Either Dopamine 5-20 mcg/kg/mt or norepinephrine 0.5-1 mcg/mt.

2.ARRYTHYMIAS:

Patients should be put on 24 hour holter monitoring. Continous monitoring should be done for development of arrythmias. Sinus tachycardia and ST depressions will revert back once the toxins are got washed out from circulation. But atrioventicular arrythmias especially ventricular tachycardia should be treated promtly. Pharmaceutical cardioversion using amiodarone can be done in hemodynamically stable patients .DC cardioversion should be given for patients with arrythmias with hemodynamic unstability.

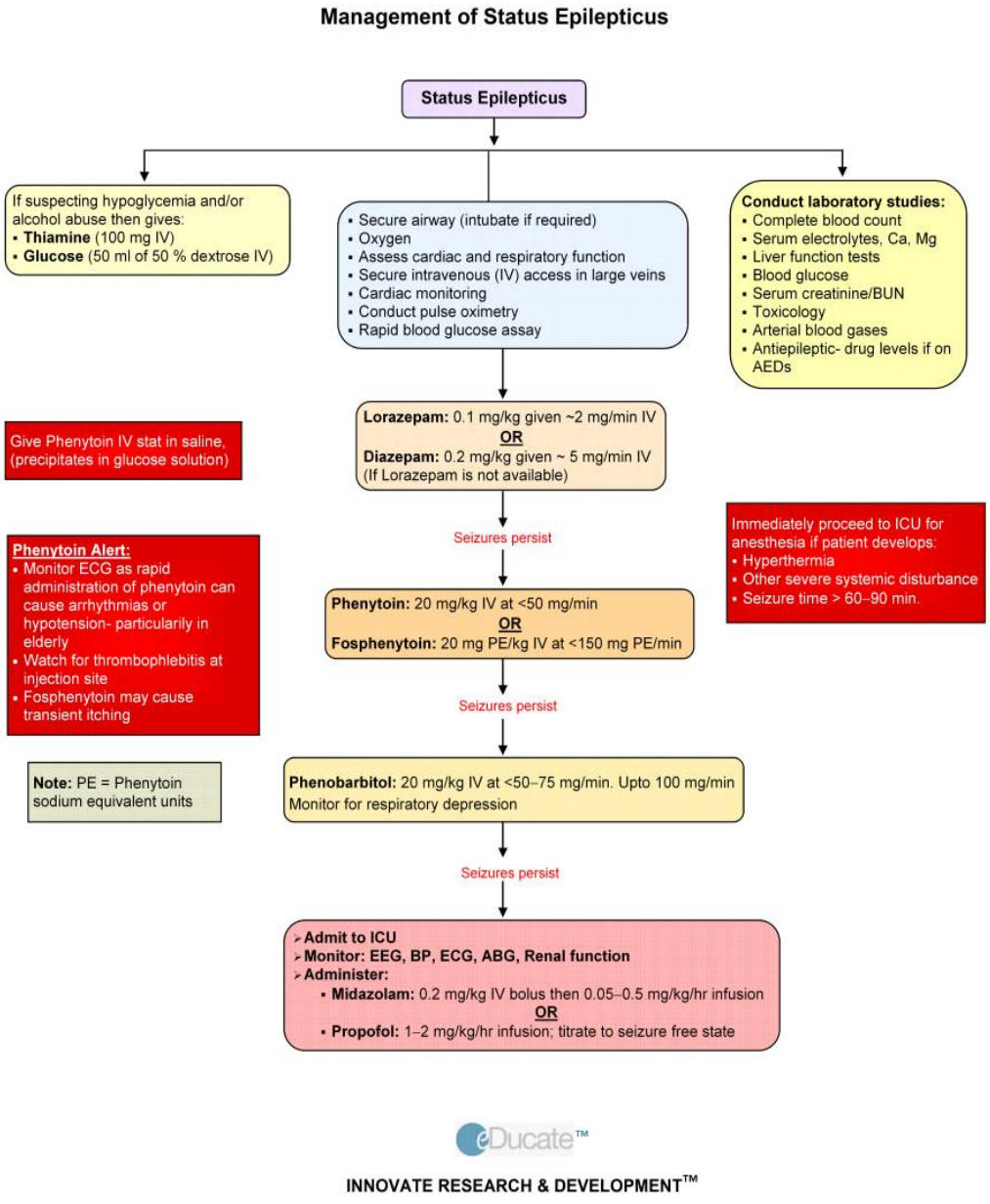


ECG OF A PATIENT SHOWING LEFT BUNDLE BRANCH BLOCK

3.SEIZURES:

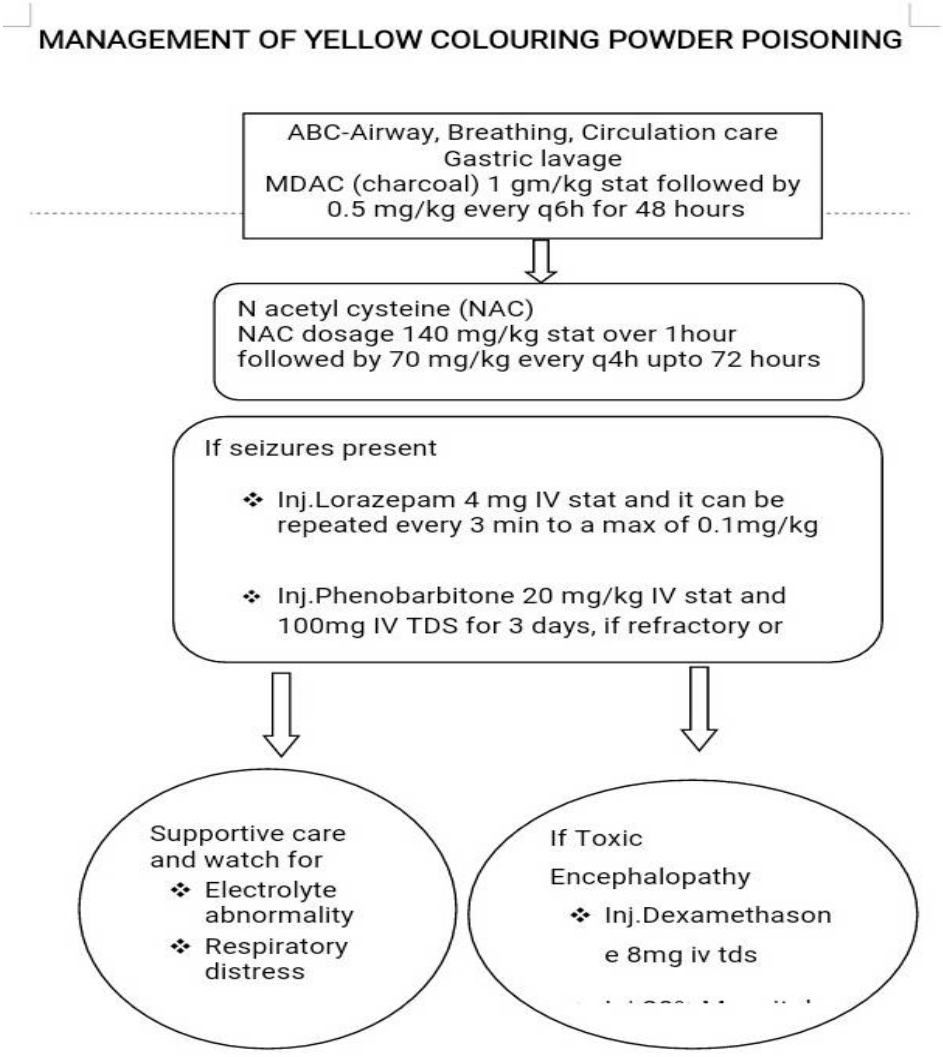
Phenytoin is contraindicated in patients with yellow cowdung coloring agent poison. The toxin combines with phenytoin causing cardiac depression.Benzodiazepines should be adminitered with caution due to

respiratory depression.diazepan can be given 5-10 mg IV repeated every 20 minutes as needed. Or lorazepam 2-4 mg initially and repeated every 20 minutes.If seizure is reccuring phenobarbotone or propofol can be given.



III. SPECIFIC TREATMENT

Anti dote is defined as a therapeutic substance used to counter act the toxic actions of a specific agent. No specific anti dote is available for yellow cow dung coloring agent poisoning. N-Acetyl cysteine is having promising results in this poisoning, because of the same toxicity pattern as acetaminophen.



MATERIALS AND METHODS

STUDY DESIGN: cross-sectional study

INCLUSION CRITERIA

Patients who have a history of exposure to yellow cow dung compound poisoning brought to the hospital within 24 hours of consumption n as indicated by patient themselves or their relatives or the doctor referring. The characteristic clinical manifestations of yellow cow dung compound poisoning should be present along with physical evidence of the poison consumed.

EXCLUSION CRITERIA

1. Patients with yellow cow dung poison mixed with any other poison.
2. Patients with history of recent Coronary artery disease.
3. Patients with a known history of renal disease and chronic liver disease.
4. Patients with history of malignancy in the past.
5. Pregnant women.
6. Patients with known history suggestive of pancreatitis.
7. Patients with muscle disorders.

METHODOLOGY

This study was conducted in the medicine department of Coimbatore Medical College Hospital with a history suggestive of yellow cow dung coloring agent poisoning during the time period of July 2016 to June 2017. A total of three hundred patients were included in our study after fulfilling the inclusion as well as exclusion criteria. The Ethical committee approval of the hospital was obtained to carry out the study in the hospital. Information was collected through a preformed questionnaire from every patient in our study. Those patients qualifying were subjected for a history, clinical examinations and biochemical investigations.

- The clinical history included the history of consumption of yellow cow dung coloring poison and presentation to our hospital within 24 hours of consumption. The patients were graded as Mild, Moderate and Severe according to the examination and clinical findings based on APACHE II scoring system. Cases were classified into mild, moderate and severe poisoning according to APACHE II severity.

- Blood investigations were collected prior to starting of the treatment. The prognostic significance of each of these parameters

is then studied in accordance with the APACHE II Score. A detailed history including Time of consumption Amount of consumption, Symptoms at presentation Pre existing medical illness were asked.

- A clinical examination including GCS, BP, SPO2, HR, RR were assessed.
- A biochemical examination including CBC, RFT, LFT, ABG, LDH, Electrolytes and ECG were done.
- Severity of poisoning was graded according to APACHE II scoring system.

Data was made into a master sheet in Microsoft excel and later converted to SPSS Software version 21.0. Student t-test was used to analyze the quantitative data. Chi square test was used to analyze qualitative data. By using these tests, the level of significance of each of these parameters was calculated. A p- value of < 0.05 is taken as statistically significant.

SOURCE OF SUBJECTS

Those patients admitted in the medical emergency department of Coimbatore Medical College and Hospital with a history of yellow cow dung coloring agent poisoning are included in our study.

SOURCE OF DATA

Data is collected by the principle investigator himself from the patients admitted with alleged history of yellow cow dung coloring agent poisoning in Coimbatore Medical College and Hospital.

DURATION OF STUDY

July 2016 to June 2017

RESULTS

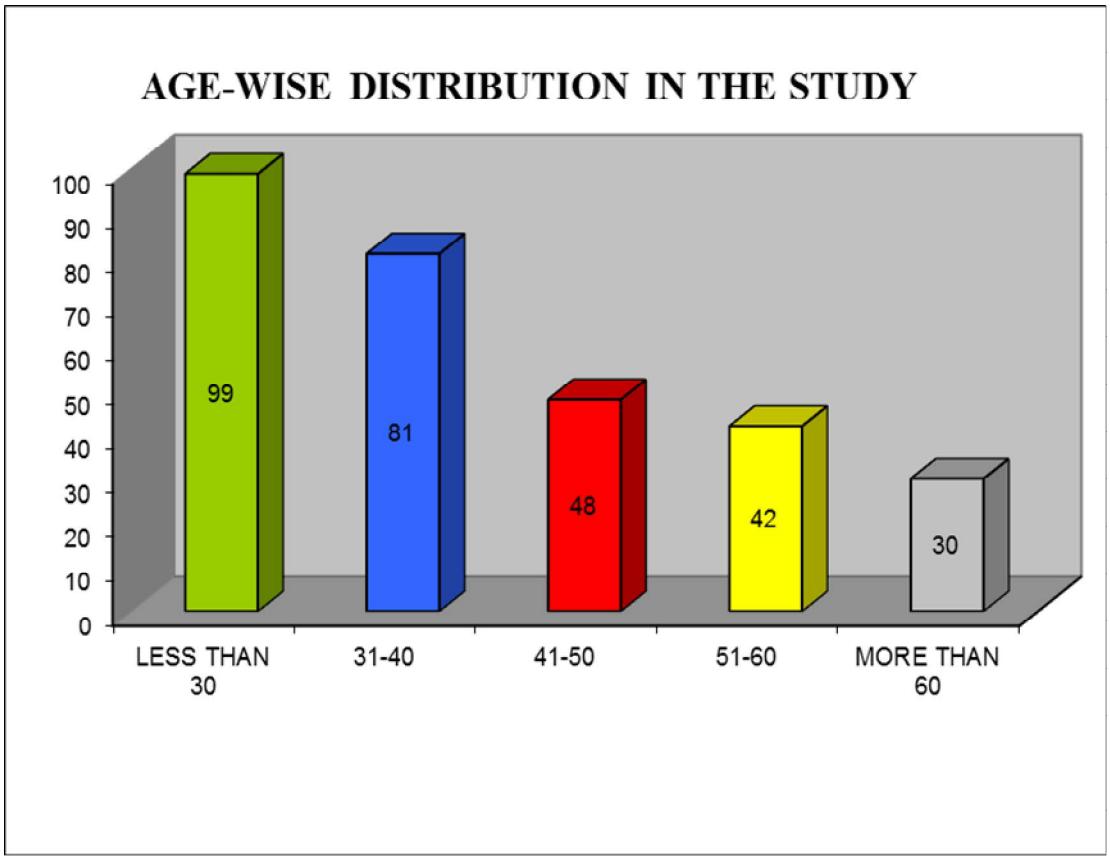
Our study population consisted of a total of 300 patients who fulfilled the inclusion and exclusion criteria. The prognostic significance of each of these values is analyzed.

The Age distribution of the study population is depicted below. Most of the cases were in the age group of less than 30 years age group.

Table-1. AGE-WISE DISTRIBUTION IN THE STUDY

AGE GROUP	NUMBER
< 30	99
31-40	81
41-50	48
51-60	42
> 60	30

Chart 1. AGE-WISE DISTRIBUTION IN THE STUDY



The Sex distribution is showed in Table-2.

Table-2 SEX DISTRIBUTION IN THE STUDY

SEX	NUMBER
MALE	111
FEMALE	189

Out of 300 patients, 111 (37%) were male and the remaining 189 (63%) were female. There was female preponderance in our study.

Chart 2. SEX DISTRIBUTION IN THE STUDY

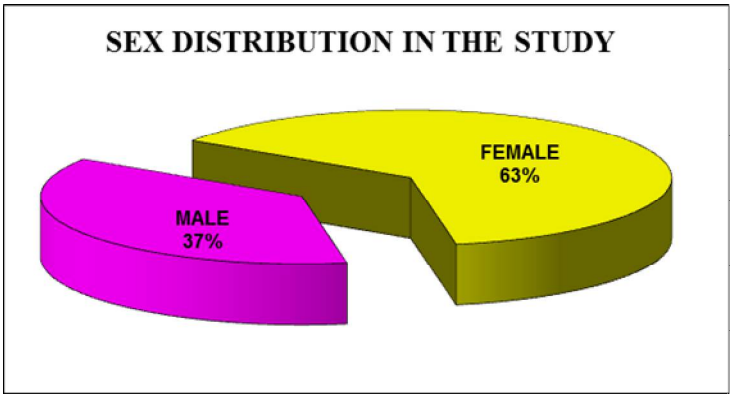


Table-3 GRADING IN ACCORDANCE WITH APACHE II SCORE

APACHE II SCORE	NUMBER OF CASES
MILD	183
MODERATE	72
SEVERE	45

Patients were graded according to APACHE II score as mild , moderate and severe. Most of the patients fell into the category of mild severity (61%). 24 % of patients were of moderate APACHE II severity score and the remaining 15 % were of severe APACHE II severity.

Chart 3. GRADING IN ACCORDANCE WITH APACHE II SCORE

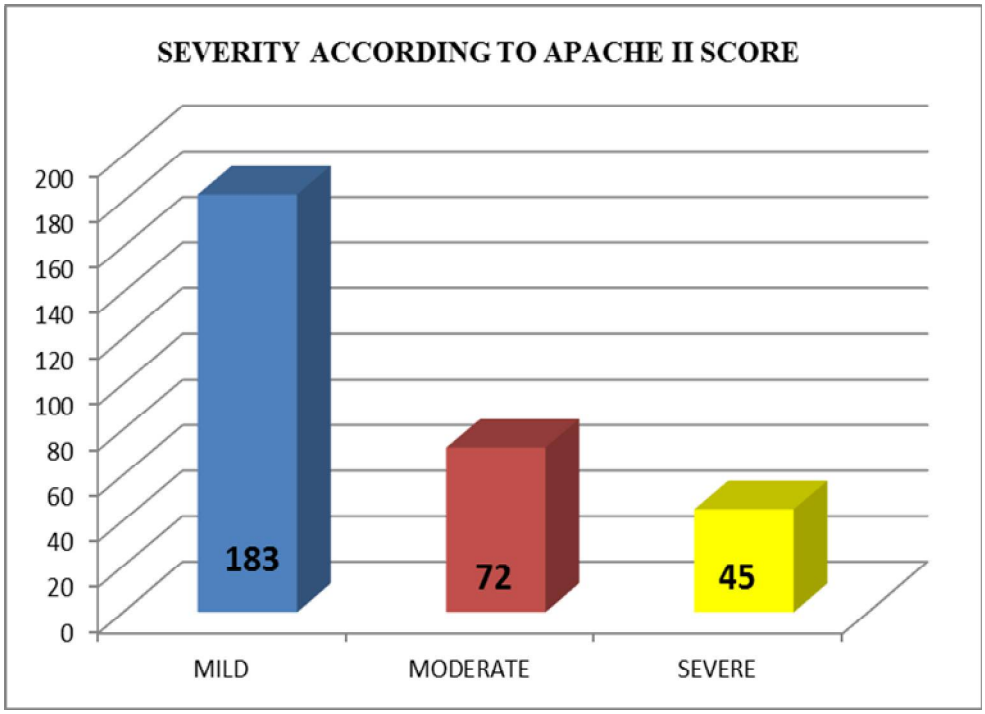


Table-4 PROGNOSIS OF THE STUDY

PROGNOSIS	PERCENTAGE
SURVIVED	281
DIED	19

In our sample study, the mortality was 6.3 %. Most of them were of APACHE II severity at the time of admission. Out of the total sample size of 300 patients, 19 patients died and 281 patients survived.

Chart-4 PROGNOSIS OF THE STUDY

The prognosis of our study is depicted in the chart below

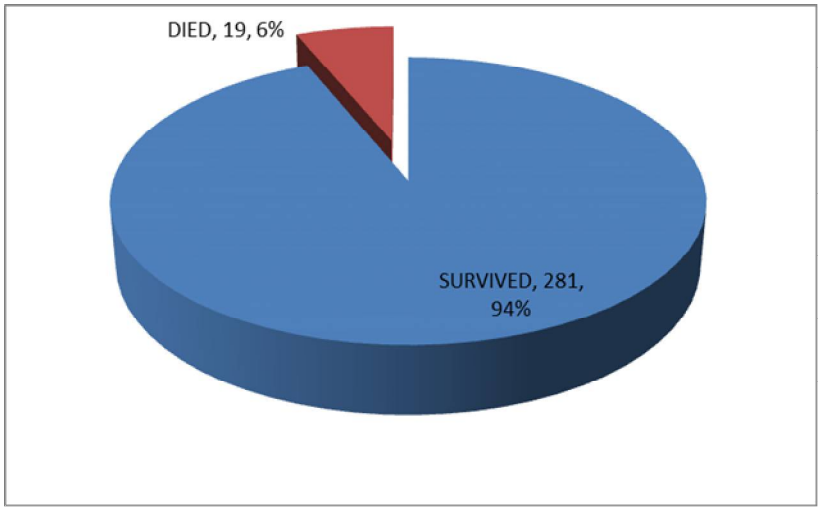
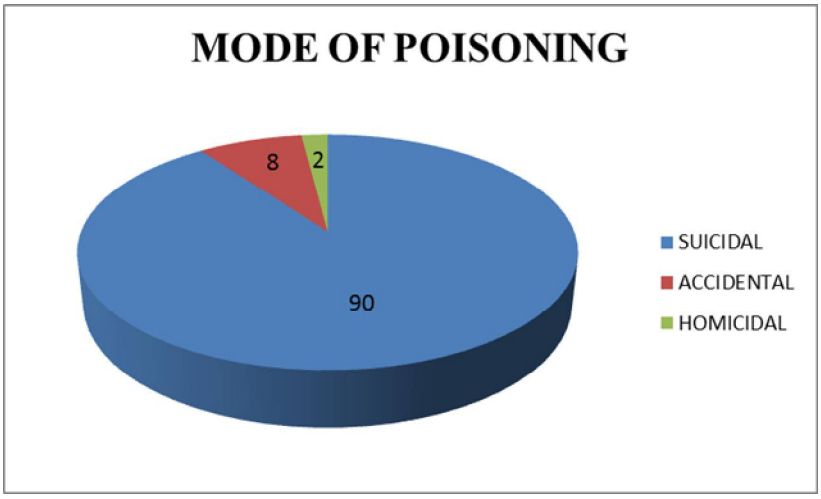


Table 5 : TABLE SHOWING MODE OF POISONING

MODE OF POISONING	NUMBER
SUICIDAL	270
ACCIDENTAL	24
HOMICIDAL	6

Chart-5 MODE OF POISONING.



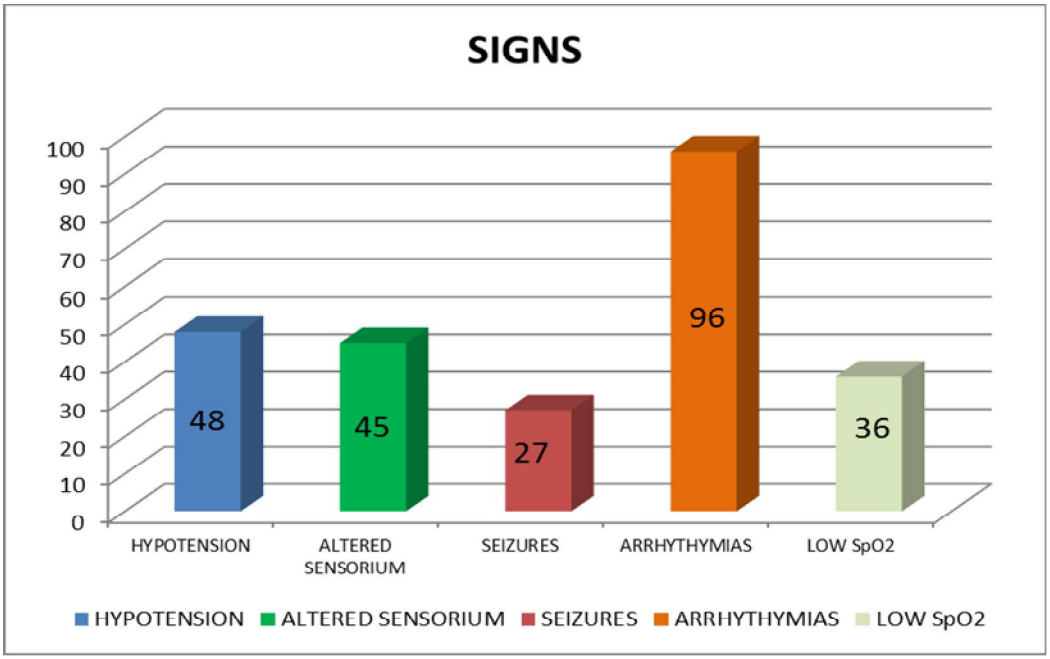
The most common intention of poisoning was suicidal (90%) followed by accidental (8%) and the least common was homicidal which was seen in 2 %.

Table 6: SIGNS OF PATIENTS AT THE TIME OF PRESENTATION

SIGNS	NUMBER OF PATIENTS
HYPOTENSION	48
ALTERED SENSORIUM	45
SEIZURES	27
ECG ABNORMALITY	96
HYPOXIA	36

This table shows the most common signs of presentation of the patient who consumed yellow coloring agent poisoning. ECG abnormality (32%) was seen in maximal number of patients followed by hypotension (16%), altered sensorium (15%), hypoxia (12 %).

Chart-6 CLINICAL SIGNS AT THE TIME OF PRESENTATION.



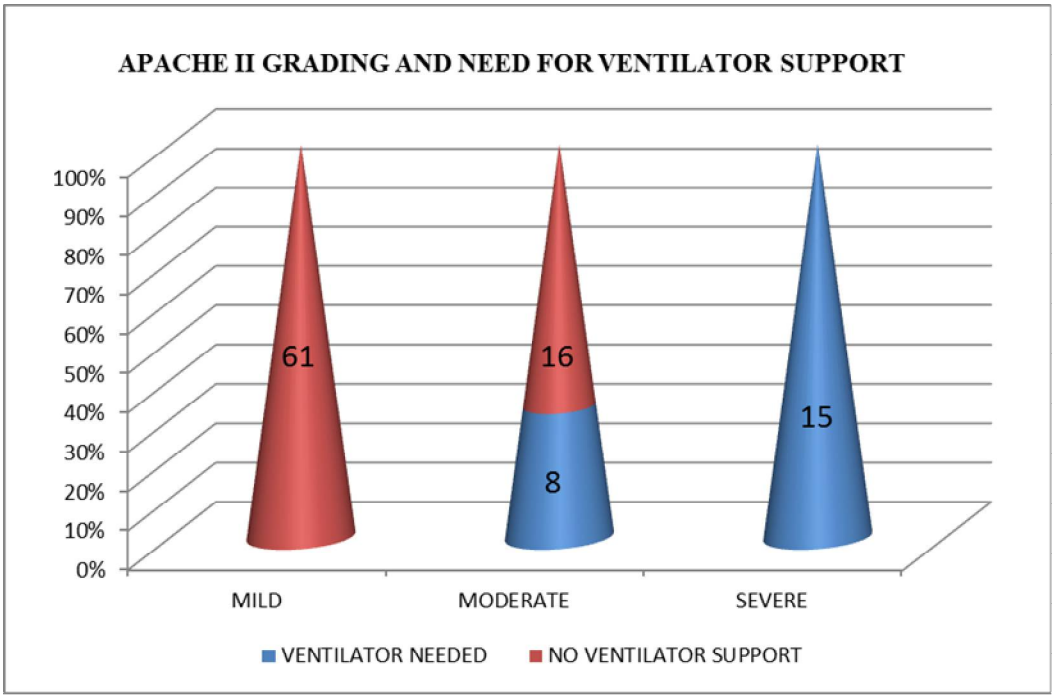
This chart depicts the clinical signs of the patient at the time of admission.

Table 7 – APACHE II grading and the need for Ventilator support

APACHE II SCALE	VENTILATOR SUPPORT		NUMBER OF PATIENTS
	YES	NO	
MILD	0	183	183
MODERATE	24	48	72
SEVERE	45	0	45

This table shows the comparison between the APACHE II score and the need for ventilator support. It was seen that out of 183 patients belonging to mild APACHE II severity, none of the patients needed ventilator support. Out of 72 patients belonging to moderate APACHE II category, 24 patients (33.3%) needed ventilator support. Whereas out of 45 patients belonging to Severe APACHE II score, all the patients needed ventilator support. This shows that the need for ventilator support has a positive corelation with the severity of APACHE II severity score. This was statistically significant $p < 0.01$.

Chart 7 – APACHE II grading and the need for Ventilator support



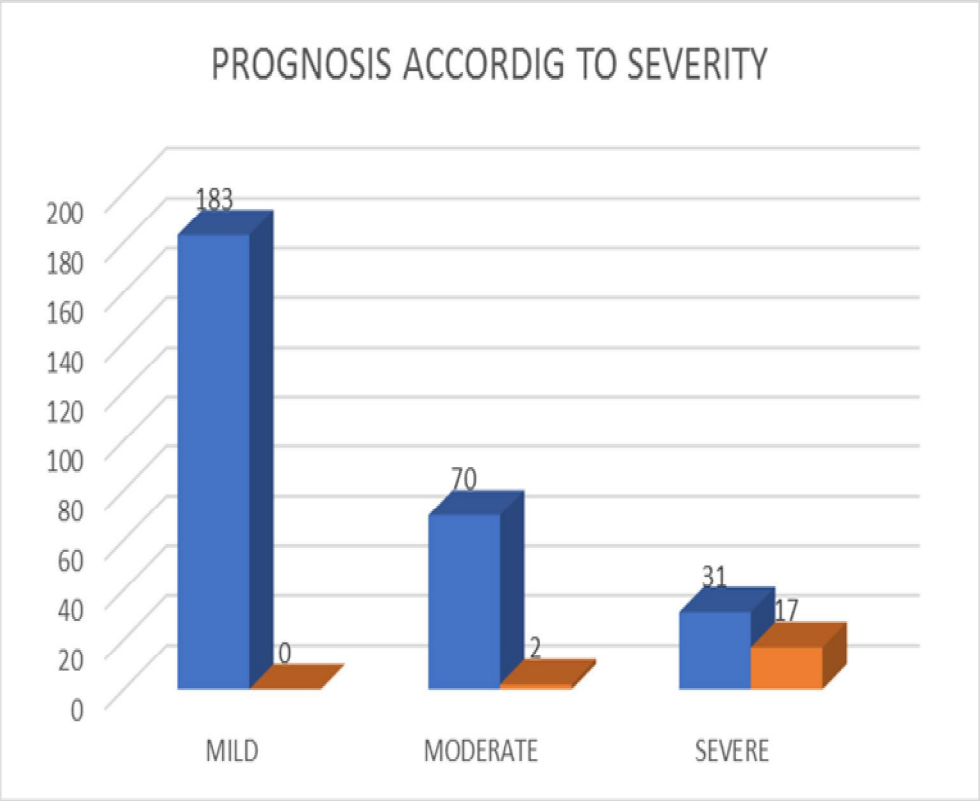
This chart shows the severity of APACHE II score and the need for Ventilator support.

TABLE 8 – PROGNOSIS ACCORDING TO SEVERITY.

APACHE II SCALE	SURVIVED	DIED	NUMBER OF PATIENTS
	YES	NO	
MILD	183	0	183
MODERATE	70	2	72
SEVERE	31	17	48

This table shows the comparions between the APACHE II score and the out come in the means of mortality. 17 out of 19 deaths were of APACHE II severity grade SEVERE.

Chart 8 – PROGNOSIS ACCORDING TO SEVERITY.



Out of the 183 cases presented with mild poisoning none of them needed ventilator care. Out of 72 cases with moderate poisoning 24 needed ventilator care, the 45 cases with severe toxicity all of them needed ventilator care.

Table 9 – APACHE II PARAMETER AND THE NEED FOR VENTILATOR SUPPORT

APACHE II SCALE PARAMETER		VENTILATOR SUPPORT		NUMBER OF PATIENTS	SIGNIFICANCE	
		YES	NO		X^2	P value
HYPOTENSION	<90SBP	36	12	48	46.401	0.001
	>90	12	240	252		
ALTERED SENSORIUM	YES	39	6	45	29.401	0.001
	NO	45	210	255		
SEIZURES	YES	27	0	27	18.840	0.001
	NO	27	246	183		
HYPOXIA	SpO2 < 90	33	3	36	31.425	0.001
	>90	24	240	264		
ECG ABNORMALITY	YES	45	51	96	32.620	0.001
	NO	24	180	204		

This table is based on the clinical sign or parameter and the need for ventilator support. Patients were classified based on the parameter at the time of admission. The parameters that were taken for computing were clinically and statistically significant. The parameters considered were Hypotension, Altered sensorium, Seizures, Hypoxia and ECG abnormality.

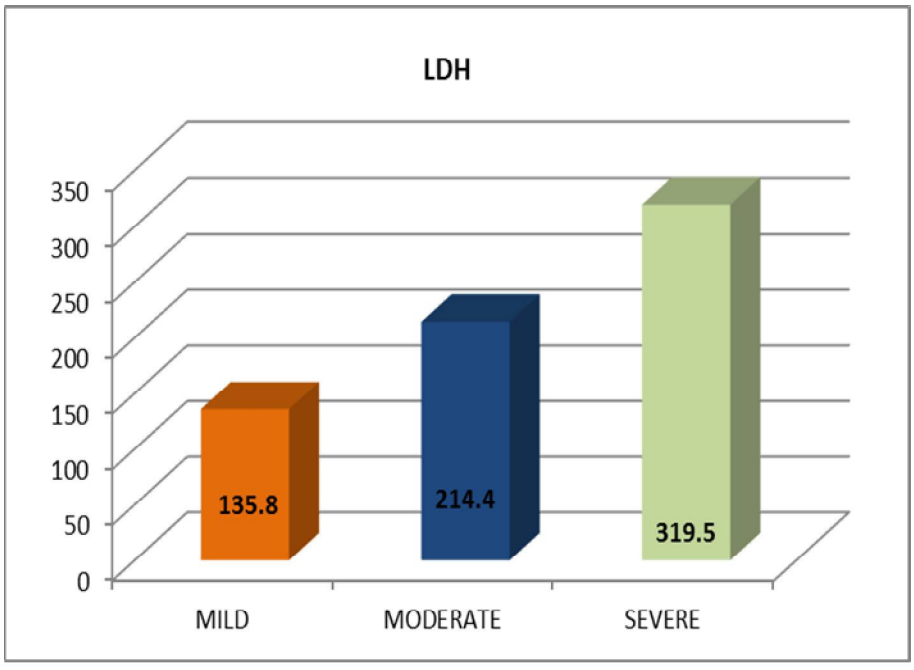
These parameters were compared with the outcome by assessing the need for ventilator support. The chi square value after analyzing in SPSS software was statistically significant ($p < 0.05$). Hence these clinical signs and parameters were having positive outcome with the severity of yellow coloring agent poisoning.

Table 10 – APACHE II grading and the average serum LDH level

APACHE II SCORE	MEAN LDH
MILD	135.8
MODERATE	214.4
SEVERE	319.5
P VALUE	0.001
SIGNIFICANCE	SIGNIFICANT

APACHE II severity was compared with the serum LDH level. There were studies that showed the positive correlation between serum LDH and the toxicity of yellow coloring agent poisoning. In patients with mild APACHE II severity, the average serum LDH level was 135.8 mg/dl and in patints with moderate severity the average LDH level was 214.4 mg/dl and the severe category was 319.5 mg/dl. Thus serum LDH level has a positive correlation ($p < 0.001$) with the severity of yellow coloring agent poisoning

Chart 10 – APACHE II grading and the average serum LDH level



This chart shows the average serum LDH level in different categories of APACHE II severity.

Table 11 – APACHE II grading and the average blood Urea level

APACHE II SCORE	MEAN BLOOD UREA
MILD	18.24
MODERATE	28.96
SEVERE	58.46
P VALUE	0.003
SIGNIFICANCE	SIGNIFICANT

Mean blood urea in mild poisoning was 18.24mg/dl whereas in severe poisoning was 58.46mg/dl which was found to be significant with a p value of 0.003. The average blood urea level was estimated in different categories of poisoning, It was seen that as the severity increases, the average blood urea level also increased.

Chart 11- Correlation between blood urea and severity of poisoning

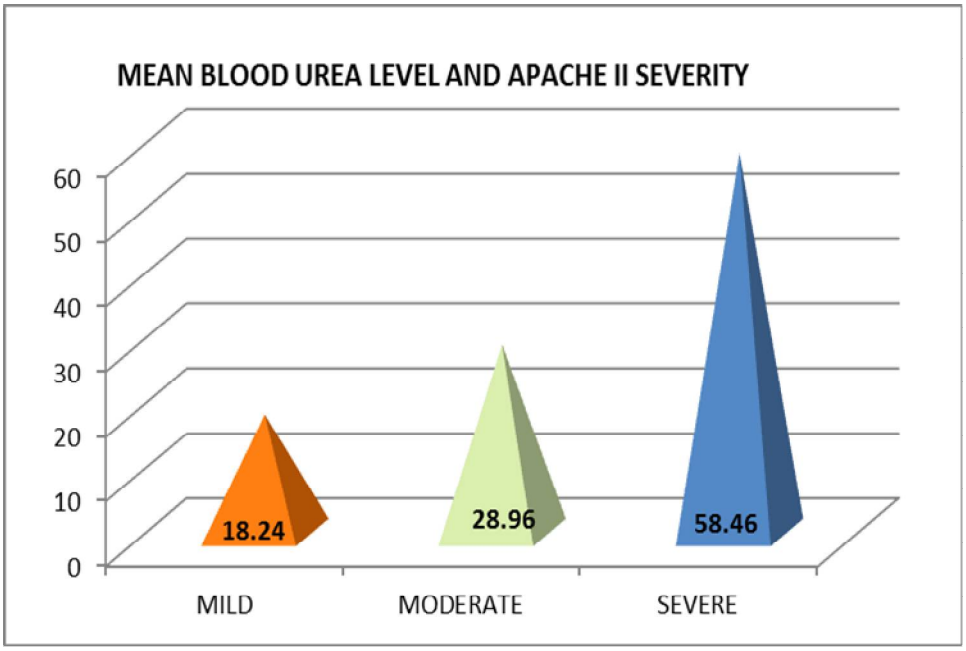
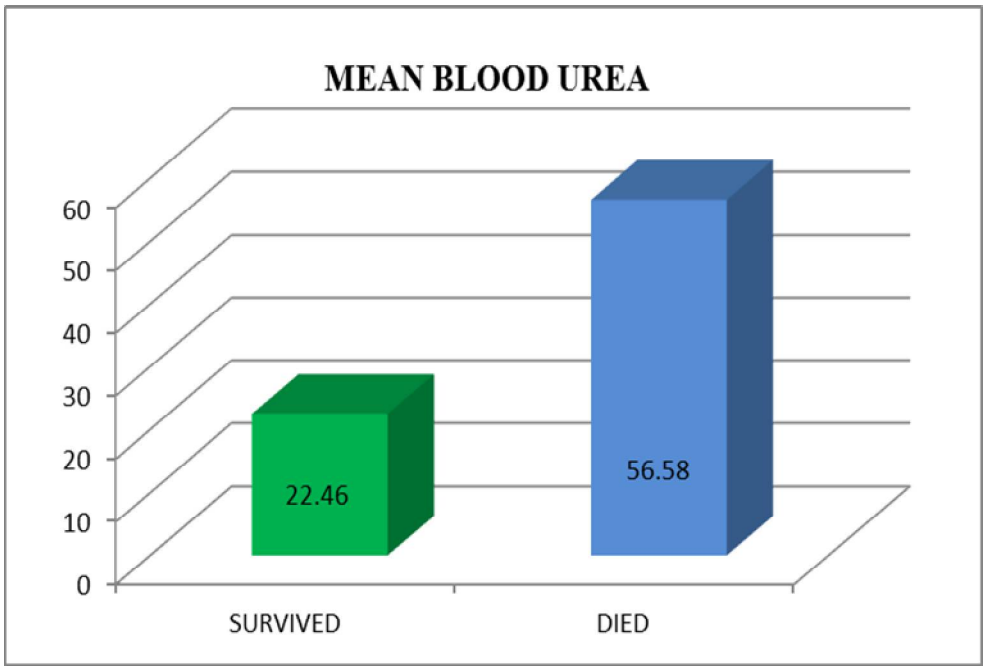


Chart 11- Average blood urea level in survived and died patients

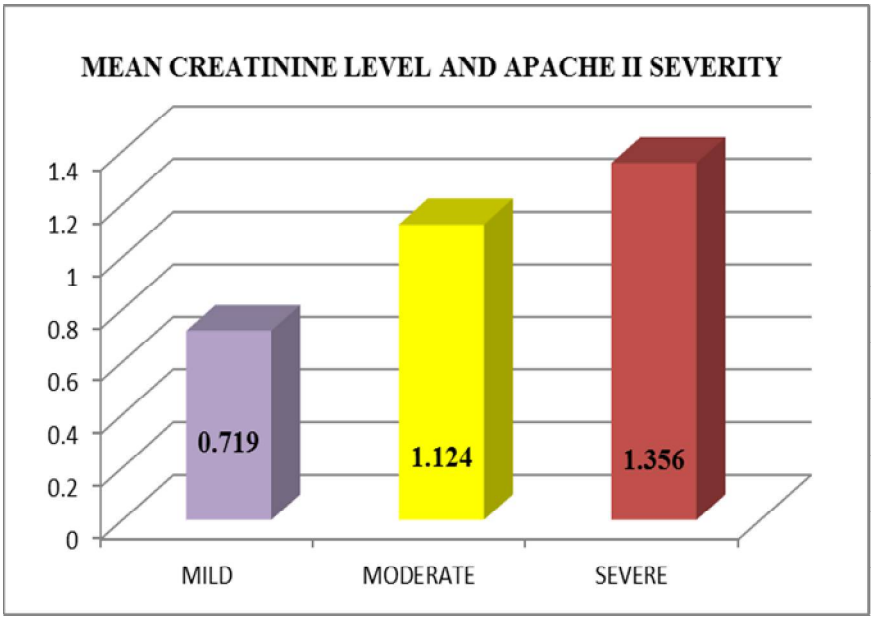


Mean serum creatinine in the study population among mild poisoning was 0.719mg/dl and in severe poisoning was 1.356mg/dl and was found to be significant at p of 0.008

Table 12- Correlation between serum creatinine and severity of poisoning

APACHE II SCORE	MEAN SERUM CREATININE
MILD	0.719
MODERATE	1.124
SEVERE	1.356
P VALUE	0.008
SIGNIFICANCE	SIGNIFICANT

CHART 12- Correlation between serum creatinine and severity of poisoning



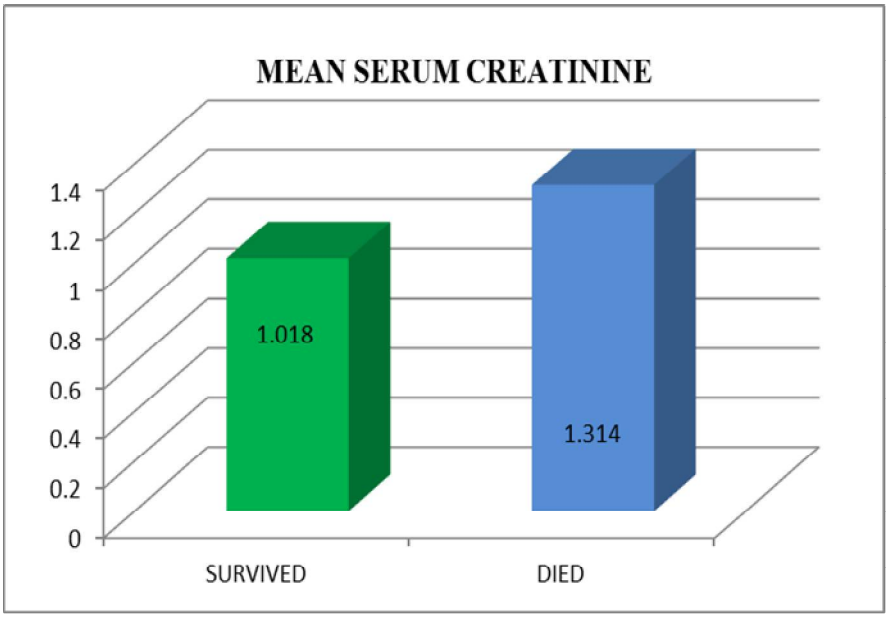
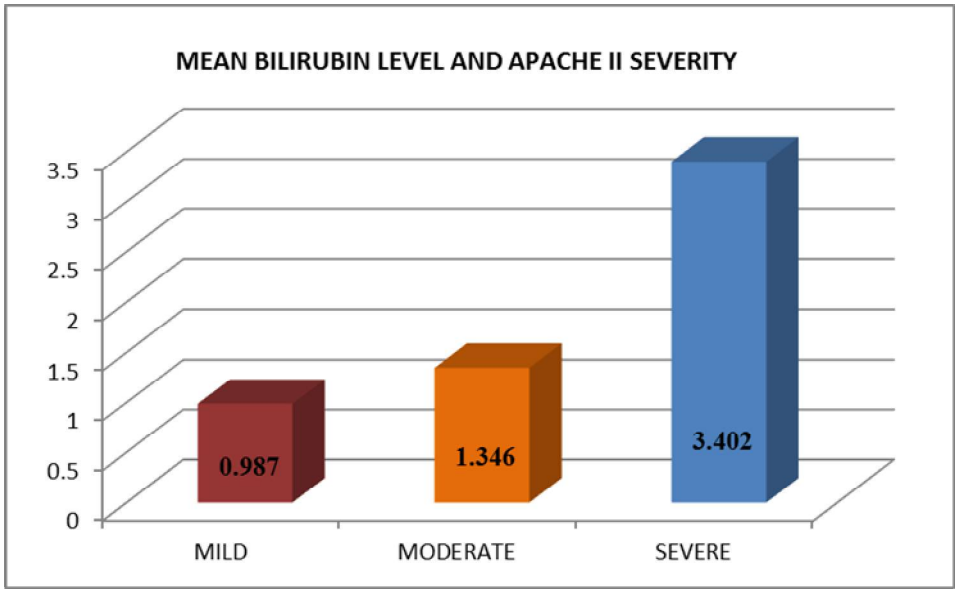


Table 13- Correlation between serum bilirubin and severity of poisoning

APACHE II SCORE	MEAN BILIRUBIN
MILD	0.987
MODERATE	1.346
SEVERE	3.402
P VALUE	0.004
SIGNIFICANCE	SIGNIFICANT

Chart 13- Correlation between serum bilirubin and severity of poisoning



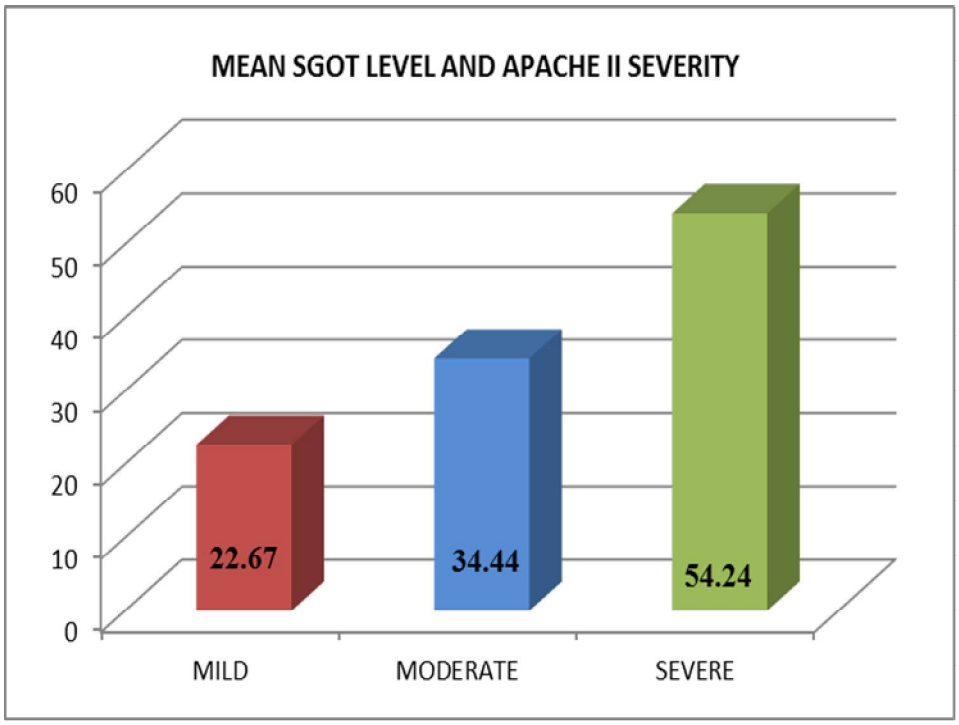
Mean SGOT level in the study population among mild poisoning was 22.67IU/L and in severe poisoning was 54.24 U/L and was found to be significant at p value of 0.001

Table 14- Correlation between SGOT and severity of poisoning

APACHE II SCORE	MEAN SGOT
MILD	22.67
MODERATE	34.44
SEVERE	54.24
P VALUE	0.001
SIGNIFICANCE	SIGNIFICANT

The mean SGOT in the survival population was 54.6 when compared to the survival population 28.55. it was statistically significant $p < 0.001$.

Chart 14. Correlation between SGOT with severity of poisoning.



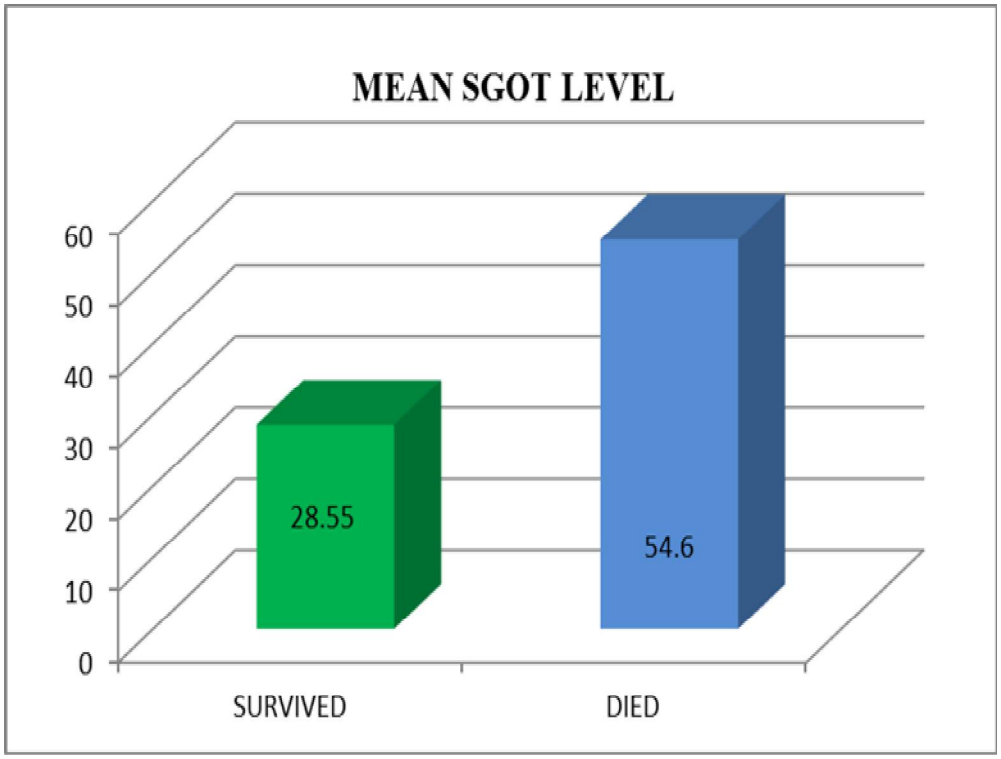
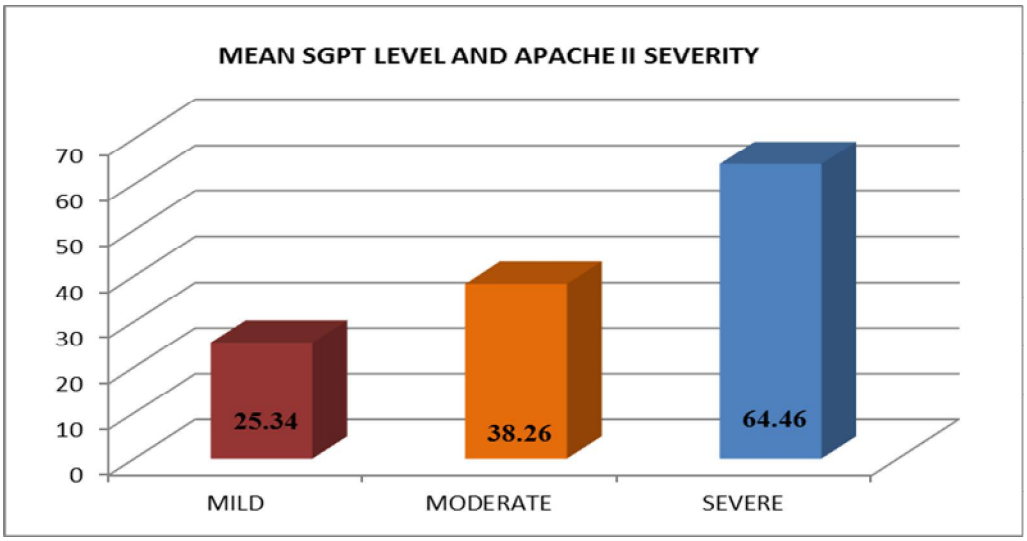


Chart 15- Correlation between SGPT levels and severity of poisoning



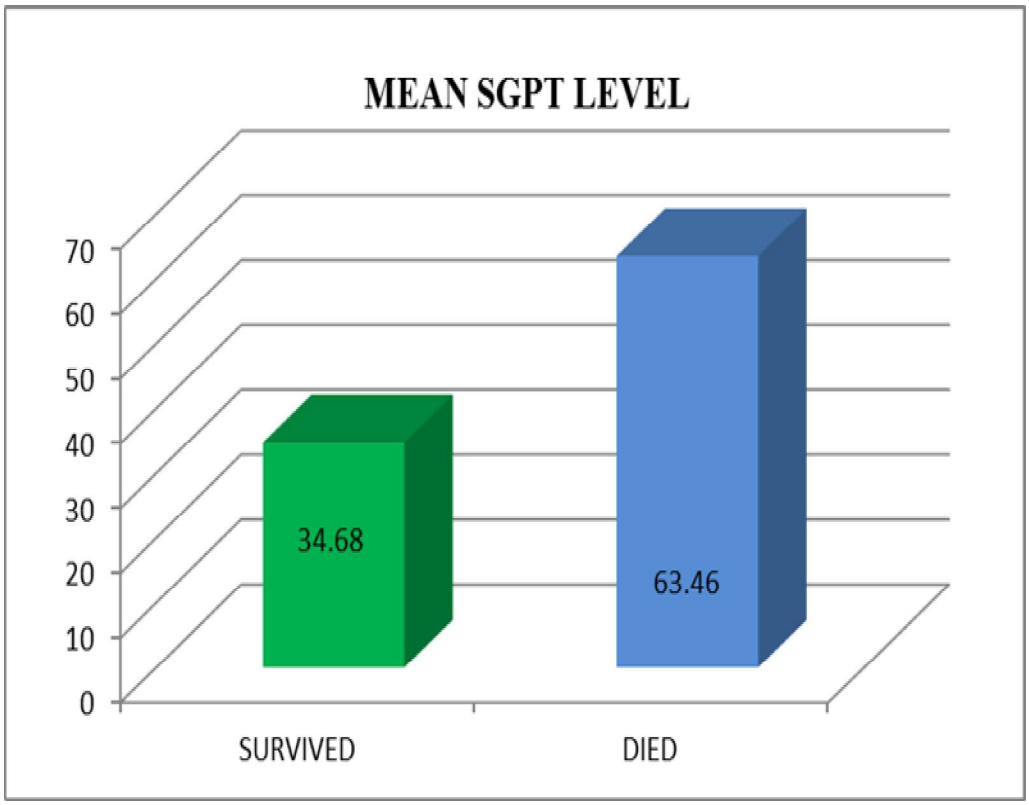


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Table 15. Correlation between SGPT levels and severity of poisoning

APACHE II SCORE	MEAN SGPT
MILD	25.34
MODERATE	38.26
SEVERE	64.46
P VALUE	0.003
SIGNIFICANCE	SIGNIFICANT

Mean SGPT level in mild poisoning was 25.34 IU/L whereas in severe poisoning was 64.46 IU/L which was found to be significant with a p value of 0.003.

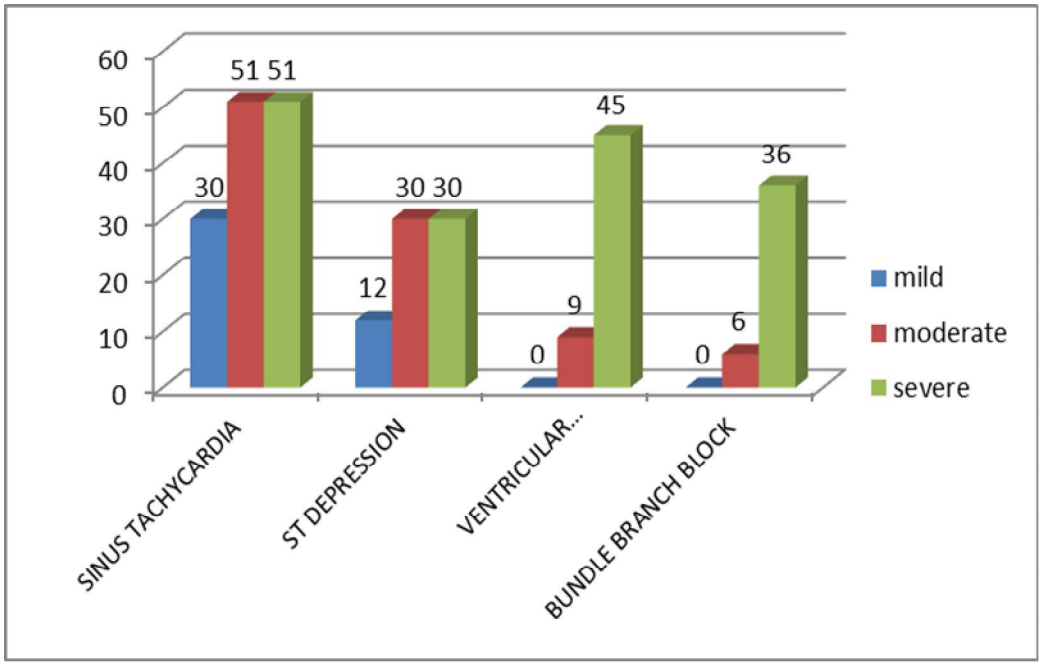
Mean SGPT level in survival population was 34.68 IU/L whereas in cases who died it was 63.46 IU/L which was found to be significant

Table 16 - Electrocardiographic findings of yellow coloring agent poisoning

ECG ABNORMALITY	MILD	MODERATE	SEVERE
SINUS TACHYCARDIA	30	51	51
ST DEPRESSION	12	30	30
VENTRICULAR TACHYARRYTHMIA	0	9	45
BUNDLE BRANCH BLOCK	0	6	36

This table shows the common ECG abnormalities in yellow coloring agent poisoning. Sinus tachycardia was the most common ECG abnormality seen in 43 % of patients. ST depression was seen in 24 % of patients. Ventricular tachycardia was seen in 18% of patients and Bundle branch block seen in 14 % of patients.

Chart 16 - Electrocardiographic findings of yellow coloring agent poisoning



This chart shows the ECG abnormality in various grades of severity of yellow coloring agent poisoning. Sinus tachycardia and ST depression was seen almost with equal incidence in all three grading of yellow coloring agent poisoning. Whereas Ventricular tachyardia and Bundle branch block was seen with increased incidence in APACHE grade SEVERE and MODERATE category.

DISCUSSION

Yellow coloring agent poisoning is most common agent that is used for suicidal purposes in southern and western part of Tamilnadu. Easy availability of the poison makes it the reason for being widely consumed compared to other poisoning. Very little understanding is available regarding the toxicology of the poison and the treatment protocol. Only very few isolated case reports are available regarding the yellow coloring agent poisoning. It is a very lethal poison having central nervous system toxicity, cardio toxicity, renal toxicity and hepatic toxicity in the form of central lobular necrosis. It is highly lipophilic and easily crosses the blood brain barrier causing CNS toxicity.

In our study of 300 patients admitted with yellow coloring agent poisoning over a period of one year, the most common age group involved was less than 30 years and with female sex preponderance. Patients were classified based on the APAHE II severity and the toxicity was studied. Most of the patients (61%) were of mild toxicity. The most common clinical signs seen in those patients were hypotension, altered sensorium, hypoxia and seizures. Ventilator support was needed in 69/300 patients (23%).

Out of 300 patients 99 were <30 years,81 were 31-40 years,48 were 41-50 years,42 were 51-60 years and 30 were >30.females constitute 189(63%) patients while males were 111(37%). According to APACHE II severity 183 (61%) were having mild poisoning,72(24%) were having moderate severe poisoning and 45(15%) had severe poisoning.

Elaborating the prognosis of the study ,out of 300 enrolled into the study, 281(94%) patients survived while 19(6%) succumbed to death.270 had suicidal intake,24 had accidental intake.

Out of 300, 48 had hypotension at the time of presentation,45 had altered sensorium,27 had seizures,96 had ECG abnormalities and 36 had hypoxia.

Out of 300,183 with mild poisoning were not in need of any ventilator support.2 out of 70 cases with moderate poisoning need ventilator care and 17 out of 48 cases of severe poisoning also needed ventilator care.

Comparing our study with the study published in Journal of The Association of Physicians of India ■ Vol. 65 ■ July 2017 ,it has been found that the study was conducted in districts of Maharashtra. 25 patients presenting with confirmed H/O consumption of (Auramine-o) synthetic yellow cow dung powder poisoning were studied. Patient's

routine investigations BSL, RFT, LFT were done. CT brain was done whenever indicated.

Of the 25 cases studied 10 were males and 15 females. Three patients were <20 years,13 were within 20-40 and 9 were >40.Ten of them presented with vomiting and epigastric pain (40%), 6 had respiratory symptoms(25%), 4 had seizures(16%) ,one had arrhythmia(1%) ,and 6 were asymptomatic.

The demographic and clinical parameters of our study were comparable with this study.

The biochemical parameters that had positive correlation with the outcome of yellow coloring agent poisoning was studied. Serum LDH was significantly high in patients with severe poisoning ($p<0.001$). the average blood urea in the study population was high in severe poisoning p 0.003. the mean blood urea was also seen to be significantly higher in the patients who died when compared to the patients who survived. The mean serum creatinine was also significantly high in severe poisoning category $p <0.003$.

The mean bilirubin level in the population was studied it was 0.987 in mild, 1.346 in moderate and 3.402 in severe poisoning category. It was seen to be statistically significant p 0.001. The correlation between SGOT and the severity was also studied. The mean SGOT in the survival population was 54.6 when compared to the survival population 28.55. it was statistically significant p <0.001. The mean SGPT level was also seen to be significantly associated with the severity of poisoning p <0.003. ECG abnormality was seen in patients with all categories of poisoning. Sinus tachycardia was the most common ECG abnormality followed by ST depression, AV arrhythmias and conduction defects.

Sinus tachycardia and ST depression was seen in all categories of poisoning. Whereas ventricular tachycardia and bundle branch block was seen with higher incidence in APACHE II severe poisoning.

In comparison of our study with study published in IOSR Journal of Dental and Medical Sciences (IOSR-JDMS) Volume 16, Issue 5 Ver. VII (May. 2017) the results were as follows.

The IOSR study included 1183 patients ,the following table compares the clinical parameters in relation to our study.

Sl no	Signs and symptoms	No of patients in IOSR	No of patients in Our study
1	Altered sensorium	685 (57%)	45(15%)
2	Seizures	367 (31%)	27(9%)
3	Required ventilator support	678 (57%)	69(23%)

Total number of patient studied were 1183 and 978 survived and 205 deaths were recorded in this study. In our study total number of cases studies were 300. Out of which 281 patients survived only 19 patients died. The number of patients having altered sensorium, seizures and ventilator support were comparatively less in our study when compared to the IOSR study.

Clinical and biochemical parameters studied in this study includes ,staining of all over the body especially hands face and tongue .Nausea, vomiting, hemetemesis, melena, hematuria, high coloured urine,epigastric tenderness abdominal pain, tenesmus , cramps, diarrhea,Purpuric spots ,haemorrage, Confusion, Irritability, flopping tremors, stupor and convulsion . These parameters are also comparable with our study.

Bio chemical abnormalities,SGOT, SGPT, Serum bilirubin, elevatedAbnormal bleeding time and clotting time

Most of the cases are presenting in the rural parts of Tamilnadu where enough treatment facilities are not available, it is necessary to triage the patients as mild, moderate and severe toxicity in the first level of health care for the need of urgent referral. Since most cases are of mild poisoning ,if the patients are properly assessed there is no need for referring all the cases to tertiary care centers and these patients can be managed safely in the primary health care level.

Based on our study we found that certain clinical parameters and also biochemical parameters were significantly associated with the severity of poisoning, we designed a scoring system for triaging the patients and to assess the need for intensive care treatment.

YELLOW COWDUNG SCORING SYSTEM

CLINICAL PARAMETERS (1/5)	LABORATORY PARAMETERS (2/5)
HYPOTENSION	*S.LDH >240*
ALTERED SENSORIUM	*BLOOD UREA >40*
SEIZURES	*S.CREATININE >1.2*
ECG ABNORMALITIES	*SGOT >40*
HYPOXIA	*S.BILIRUBIN >1.3*

If any one of the clinical or any two of the laboratory parameters are present patient can be labeled as having severe poisoning.

By applying this scoring system, we can easily assess the severity of poisoning and can refer the patients to a tertiary care center if the warning signs of the poisoning are present.

It is useful in emergency care settings were prioritizing the patient is an important criterion .As APACHE II is tedious and time consuming especially in casualty settings, yellow cow dung scoring system will be a good alternative.

SUMMARY

- Majority of patients were within the age group of <30 years, 99 patients (33%).
- Females gender predominated, 189 patients (63%).
- 183 cases (61 %) had mild poisoning, 72 cases (24%) had moderate and45 cases (15%) had severe poisoning according to APACHE score.
- In our study 281 patients survived (94%) and 19 patients died (6%).
- 270 cases had suicidal poisoning, 24 had accidental poisoning and 6 had homicidal poisoning.
- 48 cases presented with hypotension, 45 had altered sensorium, 27 had seizures,96 had ECG abnormalities and 36 cases had hypoxia.
- There was significant association with Hypotension, Low GCs, Seizures, Hypoxia and ECG abnormalities with poor prognosis ($p<0.01$).
- Out of the 183 cases presented with mild poisoning none of them needed ventilator care. Out of 72 cases with moderate poisoning 24

needed ventilator care, the 45 cases with severe toxicity all of them needed ventilator care.

- Out of 183 patients who presented with mild poisoning all of them survived. Out of 48 cases presented with severe poisoning 17 succumbed to death.
- Ventricular tachycardia and seizures were the two exclusive parameters associated with poor prognosis.
- Laboratory parameters showed abnormally elevated values in those with severe APACHE II score.
- Mean Serum LDH values is 135.8 in mild poisoning while it is 319.5 in severe poisoning.
- Mean Blood urea values in mild poisoning is 18.24 while it is 58.46 in severe poisoning.
- Mean serum Creatinine values in mild poisoning are 0.719 and in severe poisoning it is 1.356.
- Mean Bilirubin values in mild poisoning is 0.987 while it is 3.402 in severe poisoning.
- Mean SGOT level in mild poisoning is 22.67 and it is 54.24 in severe poisoning.

- Mean SGPT levels in mild poisoning is 25.34 while it is 64.46 in severe poisoning.
- Out of the ECG abnormalities, sinus tachycardia was present in mild, moderate and severe poisoning, while AV arrhythmias and conduction blocks were present only in severe poisoning.

By taking significant parameters in our study we designed a new scoring system for assessing the need for intensive care at the time of presentation at the primary health care level.

Yellow cow dung coloring agent scoring system

CLINICAL PARAMETERS (1/5)	**LABORATORY PARAMETERS (2/5)**
HYPOTENSION	*S.LDH >240*
ALTERED SENSORIUM	*BLOOD UREA >40*
SEIZURES	*S.CREATININE >1.2*
ECG ABNORMALITIES	*SGOT >40*
HYPOXIA	*S.BILIRUBIN >1.3*

If any one of the clinical or any two of the laboratory parameters are present patient should be immediately referred to a higher centre .Other patients who have only minor toxicity can be effectively managed even in primary health care levels.

The new SAANI POWDER scoring system is

- Effective,
- Easy to asses.
- Less time consuming,
- And can be used in primary health care centers to prioritize the cases and for an immediate referral to higher centers. In tertiary centers they can be used to decide upon whether the patient needs an intensive care or can be managed in medical wards.

CONCLUSION

- A total of 300 patients who met the inclusion criteria were enrolled in to the study.
- Majority of patients were within the age group of 20-30 years (33%).
- Female gender predominated, 63%.
- 61 % had mild poisoning, 24% had moderate and 15% had severe poisoning according to APACHE score.
- There was significant association with Hypotension, Low GCs, Seizures, Hypoxia and ECG abnormalities with poor prognosis ($p<0.01$).
- Ventricular tachycardia and seizures were the two exclusive parameters associated with poor prognosis.
- Laboratory parameters showed abnormally elevated values in those with severe APACHE II score.
- Out of the ECG abnormalities, sinus tachycardia was present in mild, moderate and severe poisoning, while AV arrhythmias and conduction blocks were present only in severe poisoning.

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HYPOXIA	*S.BILIRUBIN >1.3*

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INFORMED CONSENT

DEPARTMENT OF GENERAL MEDICINE

COIMBATORE MEDICAL COLLEGE COIMBATORE

Principal investigator : Dr. SHADIYA C

Research guide : Dr. USHA S

Organisation : Department of General Medicine.

Informed consent : I have been invited to participate in the research project titled **TOWARDS DEVELOPING A SCORING SYSTEM FOR RISK STRATIFICATION OF YELLOW COWDUNG COLORING AGENT POISONING AND TO ASSESS THE NEED FOR INTENSIVE CARE TREATMENT**

I understand it will be answering a set of questionnaire undergo physical examination investigates and appropriate treatment. I also give consent to utilize my personal details for the study purpose and can be contacted if necessary.

I am aware that I have the right to withdraw any time which will not affect my medical care.

Name of the participant

Signature
Date

Privacy and Confidentiality

Privacy of individuals will be respected and any information about you or provided by you during the study will be kept strictly confidential.

Authorization to publish Results

Results of the study may be published for scientific purposes and/or presented to scientific groups, however you will not be identified.

Statement of Consent

I volunteer and consent to participate in this study. I have read the consent or it has been read to me. The study has been fully explained to me, and I may ask questions at any time.

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Signature /Left thumb impression Date

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Signature of witness Date

ஒப்புதல்படிவம்

பெயர் :

வயது :

பாலினம் :

முகவரி :

கோவை அரசு மருத்துவக்கல்லூரி மருத்துவமனையில் மருத்துவர் SHADIYA C தலைமையில் நடைபெறும் இந்த ஆய்வில் முழுசம்மதத்துடன் கலந்து கொள்ள சம்மதிக்கிறேன் . இந்த ஆய்வில் என்னை பற்றி விவரங்களை பாதுகாப்புடன் இந்த ஆய்வில் வெளியிட ஆட்சேபணைஇல்லை என்று தெரிவித்துக் கொள்கிறேன். எந்த நேரத்திலும் ஆய்வில் இருந்து எந்த நேரத்திலும் விலக்கிக் கொள்ளும் உரிமை உண்டு என்று அறிவேன்.

இடம்:

தேதி: கைகெயாப்பம்/ரேகை

PROFORMA

Name		Age	
Occupation		Sex	
IP No.			
Address			
		Yes	No
Clinical Symptoms	Nausea / Vomiting		
	Abdominal Pain		
	Loose Stools		
	Seizures		
	Epigastric Pain		
	High Coloured Urine		
	Breathlessness		
	Loss of consciousness		
Past History	DM/SHT/CAD etc		
Clinical Examinations	Consciousness		
	Pulse Rate		
	Respiratory rate		
	Icterus		
	Oliguria		
	Hypotension		
	Arrythmias		
	Temperature		
	Other clinical features if any		
Blood investigations	Haemoglobin		
	total count		
	Random blood Sugar		
	B.Urea		
	S.Creatinine		
	Bilirubin T		
	SGOT		
	SGPT		
	LDH		
APACHE II Score			

KEY FOR MASTER CHART

RBS	-	Random Blood Sugar
LDL	-	Low Density Lipoprotein
VLDL	-	Very Low Density Lipoprotein
HDL	-	High Density Lipoprotein
LDH	-	Lactate Dehydrogenase
CPK	-	Creatine Phosphokinase
APACHE II	-	Acute physiology and chronic health evaluation II
SGOT	-	Serum Glutamic Oxaloa cetic Transaminase
SGPT	-	Serum Glutamic Pyruvic Transaminase
ICU	-	Intensive Care Unit
S	-	Suicidal
H	-	Homicidal
A	-	Accidental
D	-	Died
Y	-	Present
N	-	Absent
1	-	Mild
2	-	Moderate
3	-	Severe

Sl.No	NAME	AGE	SEX	MODE	APACHE 2	BP <100/60	BP 120/80	GCS<15/15	GCS15/15	SEIZURES	HYPOXIA	ECG SINUS TACHY	ST DEPRESSION	ARRYTHMIA	UREA	CREATININE	SGOT	SGPT	LDH	VENTILATION	OUTCOME
1	ASWIN	23	M	S	3	Y	N	Y	N	Y	Y	N	Y	Y	65	1.9	76	88	444	Y	D
2	VIJAYA	28	F	S	3	Y	N	Y	N	Y	Y	N	Y	Y	69	2	88	98	389	Y	D
3	MANEESHA	29	F	S	1	N	Y	N	Y	N	N	Y	N	N	35	0.7	23	18	135	N	S
4	CHELLADURAI	35	M	S	1	N	Y	N	Y	N	N	Y	N	N	33	1	22	34	140	N	S
5	MAHALAKSHMI	62	F	S	1	N	Y	N	Y	N	N	Y	N	N	39	1.1	23	36	156	N	S
6	YASODARA	39	F	S	1	N	Y	N	Y	N	N	Y	N	N	35	0.8	24	22	153	N	S
7	KHALEEL	45	M	S	1	N	Y	N	Y	N	N	Y	N	N	33	0.8	26	33	150	N	S
8	SELVI	33	F	S	2	N	Y	N	Y	Y	Y	Y	Y	N	29	1	22	15	132	Y	S
9	KALPANA	59	F	A	1	N	Y	N	Y	N	N	N	Y	N	26	0.8	36	22	146	N	S
10	SIVASAMPATH	48	M	S	2	N	Y	N	Y	N	N	N	N	N	35	0.7	24	20	220	Y	S
11	RESHMA	36	F	S	1	N	Y	N	Y	N	N	N	N	N	32	0.8	18	23	152	N	S
12	BHAGAVATHY	67	F	S	1	N	Y	N	Y	N	N	Y	N	N	46	0.8	53	42	156	N	S
13	PALANISAMY	56	M	S	1	N	Y	N	Y	N	N	Y	N	N	26	0.8	25	42	180	N	S
14	NALLAMMAL	43	F	S	1	N	Y	N	Y	N	N	N	N	N	33	0.9	23	66	150	N	S
15	GIRI	17	M	S	3	Y	N	Y	N	Y	Y	N	N	Y	66	2.2	78	88	450	Y	D
16	MAHESWARI	59	F	S	2	N	Y	N	Y	Y	Y	Y	Y	N	30	0.7	18	45	184	Y	S
17	SARASA	39	F	A	1	N	Y	N	Y	N	N	N	Y	N	29	0.9	45	23	156	N	S
18	KANNAN	15	F	S	2	N	Y	N	Y	N	N	N	N	N	36	1.2	38	26	150	N	S
19	ROJA	19	F	S	1	N	Y	N	Y	N	N	N	N	N	33	0.7	26	21	250	N	S
20	SRINIVASAN	42	M	S	1	N	Y	N	Y	N	N	N	N	N	45	0.8	35	30	136	N	S
21	AISHA	58	F	S	1	N	Y	N	Y	N	N	N	N	N	30	0.8	29	28	216	N	S
22	SIVADAS	41	M	S	3	Y	N	Y	N	Y	Y	N	N	Y	34	2.2	88	98	548	N	D
23	KUPPATHAL	38	F	S	2	N	Y	N	Y	N	N	N	Y	N	88	0.9	30	28	320	Y	S
24	SUBBAIAH	74	F	S	2	N	Y	Y	N	N	N	N	Y	N	29	1.5	26	44	227	N	S
25	SAFINA PARVEEN	56	F	S	1	N	Y	N	Y	N	N	N	N	N	42	0.9	34	32	251	N	S
26	SATHYARAJ	33	M	S	1	N	Y	N	Y	N	N	N	N	N	44	0.9	46	48	120	N	S
27	ARATHI	25	F	S	1	N	Y	N	Y	N	N	N	N	N	39	0.9	42	40	158	N	S
28	RATHNASAMY	42	M	A	1	N	Y	N	Y	N	N	N	N	N	34	1	33	32	160	N	S
29	PRIYA	36	F	S	1	N	Y	N	Y	N	N	N	N	N	33	1	35	31	194	N	S
30	SELVAM	29	M	S	1	N	Y	N	Y	N	N	N	N	N	30	0.3	38	40	120	N	S
31	BABY	53	F	S	3	Y	N	Y	N	Y	Y	N	N	Y	79	1.9	89	98	480	Y	D
32	ABUTHAHIR	49	M	S	2	N	Y	Y	N	Y	Y	N	N	N	33	0.8	48	45	201	N	S
33	MARIYAM SURAY	53	F	S	2	N	Y	Y	N	Y	Y	N	Y	N	39	1.3	52	56	324	N	S
34	SURYA	22	F	S	2	N	Y	Y	N	Y	Y	N	Y	N	29	0.9	25	22	223	Y	S
35	RAVI	47	M	S	1	N	Y	N	Y	N	N	N	N	N	32	0.7	22	20	145	N	S

Sl.No	NAME	AGE	SEX	MODE	APACHE 2	BP <100/60	BP 120/80	GCS<15/15	GCS15/15	SEIZURES	HYPOXIA	ECG SINUS TACHY	ST DEPRESSION	ARRYTHMIA	UREA	CREATININE	SGOT	SGPT	LDH	VENTILATION	OUTCOME
36	MARY	17	F	S	1	N	Y	N	Y	N	N	N	N	N	33	0.9	26	42	120	N	S
37	SENTHIL KUMAR	25	M	A	1	N	Y	N	Y	N	N	N	N	N	30	0.9	30	25	132	N	S
38	MAHESWARI	52	F	A	3	Y	N	Y	N	Y	Y	N	N	N	66	2.6	21	100	456	Y	D
39	FATHIMA	23	F	S	1	N	Y	N	Y	N	N	N	N	N	34	0.8	32	26	195	N	S
40	UMASHANKAR	44	F	S	2	N	Y	N	Y	Y	N	Y	N	N	38	1.2	36	28	218	N	S
41	LIYAKATHALI	31	M	S	2	N	Y	Y	N	Y	N	Y	N	N	50	1.6	46	42	278	N	S
42	PANDIYAMMAL	66	F	S	3	Y	N	Y	N	Y	Y	N	N	Y	63	1.9	76	102	486	Y	D
43	GOWRI	18	F	S	3	Y	N	Y	N	Y	Y	N	N	Y	56	1.9	88	112	546	Y	D
44	SANGEETHA	19	F	S	1	N	Y	N	Y	N	N	N	N	N	27	0.7	22	23	145	N	S
45	LATHAKUMARI	16	F	A	1	N	Y	N	Y	N	N	N	N	N	30	0.7	19	16	142	N	S
46	RAMPRASANTH	22	M	S	1	N	Y	N	Y	N	N	N	N	N	42	1	29	36	196	N	S
47	RAGI	15	M	H	1	N	Y	N	Y	N	N	Y	N	N	29	0.9	29	26	226	N	S
48	CHELLAMMAL	25	F	S	2	N	Y	N	Y	N	N	Y	N	N	32	0.8	24	23	156	Y	S
49	JAYASREE	36	F	S	1	N	Y	N	Y	N	N	N	N	N	39	1.4	38	22	186	N	S
50	DEVARAJ	55	M	S	3	Y	N	Y	N	Y	Y	N	N	Y	56	1.5	88	88	488	Y	D
51	SUBASHREE	39	F	S	2	N	Y	Y	N	N	Y	N	Y	N	32	0.8	39	44	235	Y	S
52	DEEPAK	41	M	S	2	N	Y	Y	N	N	Y	N	Y	N	33	0.7	22	49	163	Y	S
53	RAJI	35	F	S	1	N	Y	N	Y	N	N	N	N	N	33	0.9	51	26	156	N	S
54	SUBBU	36	M	S	2	N	Y	N	Y	N	N	N	N	N	26	0.8	45	40	355	Y	S
55	THANGAMAN	32	F	S	1	N	Y	N	Y	N	N	N	N	N	35	1	25	23	201	N	S
56	MOHANA	22	F	A	2	N	Y	N	Y	N	N	N	Y	N	25	0.9	44	40	299	Y	S
57	SAVITHA	28	F	S	1	N	Y	N	Y	N	N	N	N	N	45	1.9	39	20	201	N	S
58	VIJAYAKUMA	69	M	S	1	N	Y	N	Y	N	N	N	N	N	33	1	36	28	232	N	S
59	DEVIKA	21	F	S	1	N	Y	N	Y	N	N	N	N	N	31	0.8	28	27	220	N	S
60	SHAMIKAPOO	71	M	S	1	N	Y	N	Y	N	N	N	N	N	24	0.7	24	28	125	N	S
61	NISHANTHINI	53	F	S	1	N	Y	N	Y	N	N	N	N	N	28	0.7	20	17	185	N	S
62	BHUVANA	33	F	S	1	N	Y	N	Y	N	N	N	N	N	31	0.6	25	23	113	N	S
63	RAMACHANDR	39	M	S	1	N	Y	N	Y	N	N	Y	N	N	35	0.8	36	28	204	N	S
64	THULASI	19	F	H	1	N	Y	N	Y	N	N	Y	N	N	30	0.7	18	20	137	N	S
65	KANJAMANI	49	M	A	1	N	Y	N	Y	N	N	Y	N	N	38	1	32	29	226	N	S
66	MANJULA	36	F	S	1	N	Y	N	Y	N	N	N	N	N	31	0.9	24	19	165	N	S
67	KIRUBA	35	F	S	1	N	Y	N	Y	N	N	N	N	N	31	1	32	30	224	N	S
68	BEER MOHAM	52	M	S	1	N	Y	N	Y	N	N	N	N	N	44	1.5	42	39	294	N	S
69	DEVI	26	F	S	2	Y	N	Y	N	N	Y	Y	N	N	38	1	31	46	320	Y	S
70	SRUTHI	31	F	S	1	N	Y	N	Y	N	N	N	N	N	24	0.8	23	19	186	N	S

Sl.No	NAME	AGE	SEX	MODE	APACHE 2	BP <100/60	BP 120/80	GCS<15/15	GCS15/15	SEIZURES	HYPOXIA	ECG SINUS TACHY	ST DEPRESSION	ARRYTHMIA	UREA	CREATININE	SGOT	SGPT	LDH	VENTILATION	OUTCOME
71	ROBERT	33	M	S	1	N	Y	N	Y	N	N	N	N	N	30	0.9	53	41	321	N	S
72	SRUTHI	34	F	S	1	N	Y	N	Y	N	N	N	N	N	33	0.9	34	32	208	N	S
73	MIZAD	32	M	S	1	N	Y	N	Y	N	N	N	N	N	30	1	21	18	143	N	S
74	KULANTHIYAMMAL	31	F	S	3	Y	N	Y	N	Y	Y	N	Y	Y	55	1.9	88	96	889	Y	D
75	CHELLAM	35	F	S	1	N	Y	N	Y	N	N	N	N	N	27	0.7	22	19	164	N	S
76	SAM FRANKLIN	47	M	S	2	Y	N	Y	N	N	Y	N	Y	N	30	0.8	39	36	320	N	S
77	MAYILATHAL	51	F	S	3	Y	N	Y	N	Y	Y	N	Y	Y	89	1.1	84	90	448	Y	D
78	SARADHA	34	F	S	1	N	Y	N	Y	N	N	N	N	N	26	0.8	46	42	210	N	S
79	VINITHA	26	F	S	1	N	Y	N	Y	N	N	N	N	N	22	0.8	17	16	200	N	S
80	KALIMUTHU	32	M	S	1	N	Y	N	Y	N	N	Y	N	N	34	0.7	21	18	194	N	S
81	MEHARUNNISA	39	F	S	1	N	Y	N	Y	N	N	N	N	N	30	0.9	39	36	230	N	S
82	KRISHNA	28	F	S	3	Y	N	Y	N	Y	Y	N	N	Y	56	1.6	78	96	652	Y	D
83	BALASENTHIL	44	M	S	1	N	Y	N	Y	N	N	N	N	N	29	0.9	46	44	210	N	S
84	SANKARMANI	68	F	S	1	N	Y	N	Y	N	N	N	N	N	30	0.6	16	17	123	N	S
85	SUSIL	33	F	S	1	N	Y	N	Y	N	N	N	N	N	22	0.8	20	18	178	N	S
86	MANJULA	39	F	S	2	Y	N	N	Y	Y	Y	N	N	N	31	1	44	38	265	Y	S
87	KALAIYARASAN	29	M	S	3	Y	N	Y	N	Y	Y	N	Y	Y	88	1.9	75	98	654	Y	D
88	PAVITHRA	55	F	S	2	Y	N	Y	N	N	Y	N	N	N	45	1.2	48	39	321	N	S
89	PAVIYTHRAN	22	F	A	1	N	Y	N	Y	N	N	N	N	N	28	0.8	25	23	187	N	S
90	PREETHI	34	F	S	1	N	Y	N	Y	N	N	N	Y	N	29	0.9	36	35	245	N	S
91	SATHYA	33	F	S	2	N	Y	N	Y	N	N	N	Y	N	26	0.7	22	20	252	N	S
92	RANJITH	35	M	S	2	N	Y	N	Y	N	N	Y	N	N	33	1.2	19	39	221	N	S
93	NAVAKODEESW	46	M	S	1	N	Y	N	Y	N	N	Y	N	N	29	0.9	30	28	215	N	S
94	ANANTHAKUMA	36	F	S	3	Y	N	Y	N	Y	Y	Y	N	Y	66	1.8	78	100	662	Y	S
95	MARUTHAMMAL	69	F	S	2	Y	N	Y	N	Y	Y	N	Y	N	37	1	35	34	365	N	D
96	BHAGYALAKSH	56	F	S	2	Y	N	Y	N	Y	Y	N	Y	N	43	1.3	38	37	287	N	S
97	BALAMURUGAN	63	M	S	1	N	Y	N	Y	N	N	N	N	N	28	0.8	23	29	294	N	S
98	SIVASANKAR	47	M	S	1	N	Y	N	Y	N	N	N	N	N	30	0.8	22	26	204	N	S
99	KUPPAMMAL	58	F	S	3	Y	N	Y	N	Y	Y	N	N	Y	65	1.5	98	62	662	Y	D
100	GEORGE	49	M	S	1	N	Y	N	Y	N	N	Y	N	N	36	0.8	26	42	272	N	S
101	JAYASREE	36	F	S	1	N	Y	N	Y	N	N	N	N	N	39	1.4	38	22	186	N	S
102	DEVARAJ	55	M	S	3	Y	N	Y	N	Y	Y	N	N	Y	56	1.5	88	88	488	Y	D
103	SUBASHREE	39	F	S	2	N	Y	Y	N	N	Y	N	Y	N	32	0.8	39	44	235	Y	S
104	DEEPAK	41	M	S	2	N	Y	Y	N	N	Y	N	Y	N	33	0.7	22	49	163	Y	S
105	RAJI	35	F	S	1	N	Y	N	Y	N	N	N	N	N	33	0.9	51	26	156	N	S

Sl.No	NAME	AGE	SEX	MODE	APACHE 2	BP <100/60	BP 120/80	GCS<15/15	GCS15/15	SEIZURES	HYPOXIA	ECG SINUS TACHY	ST DEPRESSION	ARRYTHMIA	UREA	CREATININE	SGOT	SGPT	LDH	VENTILATION	OUTCOME
106	SUBBU	36	M	S	2	N	Y	N	Y	N	N	N	N	N	26	0.8	45	40	355	Y	S
107	THANGAMAN	32	F	S	1	N	Y	N	Y	N	N	N	N	N	35	1	25	23	201	N	S
108	MOHANA	22	F	A	2	N	Y	N	Y	N	N	N	Y	N	25	0.9	44	40	299	Y	S
109	SAVITHA	28	F	S	1	N	Y	N	Y	N	N	N	N	N	45	1.9	39	20	201	N	S
110	VIJAYAKUMA	69	M	S	1	N	Y	N	Y	N	N	N	N	N	33	1	36	28	232	N	S
111	DEVIKA	21	F	S	1	N	Y	N	Y	N	N	N	N	N	31	0.8	28	27	220	N	S
112	SHAMIKAPOO	71	M	S	1	N	Y	N	Y	N	N	N	N	N	24	0.7	24	28	125	N	S
113	NISHANTHINI	53	F	S	1	N	Y	N	Y	N	N	N	N	N	28	0.7	20	17	185	N	S
114	BHUVANA	33	F	S	1	N	Y	N	Y	N	N	N	N	N	31	0.6	25	23	113	N	S
115	RAMACHANDR	39	M	S	1	N	Y	N	Y	N	N	Y	N	N	35	0.8	36	28	204	N	S
116	THULASI	19	F	H	1	N	Y	N	Y	N	N	Y	N	N	30	0.7	18	20	137	N	S
117	KANJAMANI	49	M	A	1	N	Y	N	Y	N	N	Y	N	N	38	1	32	29	226	N	S
118	MANJULA	36	F	S	1	N	Y	N	Y	N	N	N	N	N	31	0.9	24	19	165	N	S
119	KIRUBA	35	F	S	1	N	Y	N	Y	N	N	N	N	N	31	1	32	30	224	N	S
120	BEER MOHAM	52	M	S	1	N	Y	N	Y	N	N	N	N	N	44	1.5	42	39	294	N	S
121	DEVI	26	F	S	2	Y	N	Y	N	N	Y	Y	N	N	38	1	31	46	320	Y	S
122	SRUTHI	31	F	S	1	N	Y	N	Y	N	N	N	N	N	24	0.8	23	19	186	N	S
123	ROBERT	33	M	S	1	N	Y	N	Y	N	N	N	N	N	30	0.9	53	41	321	N	S
124	SRUTHI	34	F	S	1	N	Y	N	Y	N	N	N	N	N	33	0.9	34	32	208	N	S
125	MIZAD	32	M	S	1	N	Y	N	Y	N	N	N	N	N	30	1	21	18	143	N	S
126	KULANTHIYAMMAL	31	F	S	3	Y	N	Y	N	Y	Y	N	Y	Y	55	1.9	88	96	889	Y	D
127	THULASI	19	F	H	1	N	Y	N	Y	N	N	Y	N	N	30	0.7	18	20	137	N	S
128	KANJAMANI	49	M	A	1	N	Y	N	Y	N	N	Y	N	N	38	1	32	29	226	N	S
129	MANJULA	36	F	S	1	N	Y	N	Y	N	N	N	N	N	31	0.9	24	19	165	N	S
130	KIRUBA	35	F	S	1	N	Y	N	Y	N	N	N	N	N	31	1	32	30	224	N	S
131	BEER MOHAM	52	M	S	1	N	Y	N	Y	N	N	N	N	N	44	1.5	42	39	294	N	S
132	DEVI	26	F	S	2	Y	N	Y	N	N	Y	Y	N	N	38	1	31	46	320	Y	S
133	SRUTHI	31	F	S	1	N	Y	N	Y	N	N	N	N	N	24	0.8	23	19	186	N	S
134	ROBERT	33	M	S	1	N	Y	N	Y	N	N	N	N	N	30	0.9	53	41	321	N	S
135	SRUTHI	34	F	S	1	N	Y	N	Y	N	N	N	N	N	33	0.9	34	32	208	N	S
136	MIZAD	32	M	S	1	N	Y	N	Y	N	N	N	N	N	30	1	21	18	143	N	S
137	KULANTHIYAMMAL	31	F	S	3	Y	N	Y	N	Y	Y	N	Y	Y	55	1.9	88	96	889	Y	D
138	CHELLAM	35	F	S	1	N	Y	N	Y	N	N	N	N	N	27	0.7	22	19	164	N	S
139	SAM FRANKLIN	47	M	S	2	Y	N	Y	N	N	Y	N	Y	N	30	0.8	39	36	320	N	S
140	MAYILATHAL	51	F	S	3	Y	N	Y	N	Y	Y	N	Y	Y	89	1.1	84	90	448	Y	D

Sl.No	NAME	AGE	SEX	MODE	APACHE 2	BP <100/60	BP 120/80	GCS<15/15	GCS15/15	SEIZURES	HYPOXIA	ECG SINUS TACHY	ST DEPRESSION	ARRYTHMIA	UREA	CREATININE	SGOT	SGPT	LDH	VENTILATION	OUTCOME
141	SARADHA	34	F	S	1	N	Y	N	Y	N	N	N	N	N	26	0.8	46	42	210	N	S
142	VINITHA	26	F	S	1	N	Y	N	Y	N	N	N	N	N	22	0.8	17	16	200	N	S
143	KALIMUTHU	32	M	S	1	N	Y	N	Y	N	N	Y	N	N	34	0.7	21	18	194	N	S
144	MEHARUNNISA	39	F	S	1	N	Y	N	Y	N	N	N	N	N	30	0.9	39	36	230	N	S
145	KRISHNA	28	F	S	3	Y	N	Y	N	Y	Y	N	N	Y	56	1.6	78	96	652	Y	D
146	BALASENTHIL	44	M	S	1	N	Y	N	Y	N	N	N	N	N	29	0.9	46	44	210	N	S
147	SANKARMANI	68	F	S	1	N	Y	N	Y	N	N	N	N	N	30	0.6	16	17	123	N	S
148	SUSIL	33	F	S	1	N	Y	N	Y	N	N	N	N	N	22	0.8	20	18	178	N	S
149	MANJULA	39	F	S	2	Y	N	N	Y	Y	Y	N	N	N	31	1	44	38	265	Y	S
150	KALAIYARASAN	29	M	S	3	Y	N	Y	N	Y	Y	N	Y	Y	88	1.9	75	98	654	Y	D
151	PAVITHRA	55	F	S	2	Y	N	Y	N	N	Y	N	N	N	45	1.2	48	39	321	N	S
152	PAVIYTHRAN	22	F	A	1	N	Y	N	Y	N	N	N	N	N	28	0.8	25	23	187	N	S
153	PREETHI	34	F	S	1	N	Y	N	Y	N	N	N	Y	N	29	0.9	36	35	245	N	S
154	SATHYA	33	F	S	2	N	Y	N	Y	N	N	N	Y	N	26	0.7	22	20	252	N	S
155	RANJITH	35	M	S	2	N	Y	N	Y	N	N	Y	N	N	33	1.2	19	39	221	N	S
156	NAVAKODEESW	46	M	S	1	N	Y	N	Y	N	N	Y	N	N	29	0.9	30	28	215	N	S
157	ANANTHAKUMA	36	F	S	3	Y	N	Y	N	Y	Y	Y	N	Y	66	1.8	78	100	662	Y	S
158	MARUTHAMMAL	69	F	S	2	Y	N	Y	N	Y	Y	N	Y	N	37	1	35	34	365	N	D
159	BHAGYALAKSH	56	F	S	2	Y	N	Y	N	Y	Y	N	Y	N	43	1.3	38	37	287	N	S
160	BALAMURUGAN	63	M	S	1	N	Y	N	Y	N	N	N	N	N	28	0.8	23	29	294	N	S
161	SIVASANKAR	47	M	S	1	N	Y	N	Y	N	N	N	N	N	30	0.8	22	26	204	N	S
162	KUPPAMMAL	58	F	S	3	Y	N	Y	N	Y	Y	N	N	Y	65	1.5	98	62	662	Y	D
163	GEORGE	49	M	S	1	N	Y	N	Y	N	N	Y	N	N	36	0.8	26	42	272	N	S
164	JAYASREE	36	F	S	1	N	Y	N	Y	N	N	N	N	N	39	1.4	38	22	186	N	S
165	DEVARAJ	55	M	S	3	Y	N	Y	N	Y	Y	N	N	Y	56	1.5	88	88	488	Y	D
166	SUBASHREE	39	F	S	2	N	Y	Y	N	N	Y	N	Y	N	32	0.8	39	44	235	Y	S
167	DEEPAK	41	M	S	2	N	Y	Y	N	N	Y	N	Y	N	33	0.7	22	49	163	Y	S
168	RAJI	35	F	S	1	N	Y	N	Y	N	N	N	N	N	33	0.9	51	26	156	N	S
169	SUBBU	36	M	S	2	N	Y	N	Y	N	N	N	N	N	26	0.8	45	40	355	Y	S
170	THANGAMAN	32	F	S	1	N	Y	N	Y	N	N	N	N	N	35	1	25	23	201	N	S
171	MOHANA	22	F	A	2	N	Y	N	Y	N	N	N	Y	N	25	0.9	44	40	299	Y	S
172	SAVITHA	28	F	S	1	N	Y	N	Y	N	N	N	N	N	45	1.9	39	20	201	N	S
173	VIJAYAKUMA	69	M	S	1	N	Y	N	Y	N	N	N	N	N	33	1	36	28	232	N	S
174	DEVIKA	21	F	S	1	N	Y	N	Y	N	N	N	N	N	31	0.8	28	27	220	N	S
175	SHAMIKAPOO	71	M	S	1	N	Y	N	Y	N	N	N	N	N	24	0.7	24	28	125	N	S

Sl.No	NAME	AGE	SEX	MODE	APACHE 2	BP <100/60	BP 120/80	GCS<15/15	GCS15/15	SEIZURES	HYPOXIA	ECG SINUS TACHY	ST DEPRESSION	ARRYTHMIA	UREA	CREATININE	SGOT	SGPT	LDH	VENTILATION	OUTCOME
176	NISHANTHINI	53	F	S	1	N	Y	N	Y	N	N	N	N	N	28	0.7	20	17	185	N	S
177	BHUVANA	33	F	S	1	N	Y	N	Y	N	N	N	N	N	31	0.6	25	23	113	N	S
178	RAMACHANDR	39	M	S	1	N	Y	N	Y	N	N	Y	N	N	35	0.8	36	28	204	N	S
179	THULASI	19	F	H	1	N	Y	N	Y	N	N	Y	N	N	30	0.7	18	20	137	N	S
180	KANJAMANI	49	M	A	1	N	Y	N	Y	N	N	Y	N	N	38	1	32	29	226	N	S
181	MANJULA	36	F	S	1	N	Y	N	Y	N	N	N	N	N	31	0.9	24	19	165	N	S
182	KIRUBA	35	F	S	1	N	Y	N	Y	N	N	N	N	N	31	1	32	30	224	N	S
183	BEER MOHAM	52	M	S	1	N	Y	N	Y	N	N	N	N	N	44	1.5	42	39	294	N	S
184	DEVI	26	F	S	2	Y	N	Y	N	N	Y	Y	N	N	38	1	31	46	320	Y	S
185	SRUTHI	31	F	S	1	N	Y	N	Y	N	N	N	N	N	24	0.8	23	19	186	N	S
186	ROBERT	33	M	S	1	N	Y	N	Y	N	N	N	N	N	30	0.9	53	41	321	N	S
187	SRUTHI	34	F	S	1	N	Y	N	Y	N	N	N	N	N	33	0.9	34	32	208	N	S
188	MIZAD	32	M	S	1	N	Y	N	Y	N	N	N	N	N	30	1	21	18	143	N	S
189	KULANTHIYAMMAL	31	F	S	3	Y	N	Y	N	Y	Y	N	Y	Y	55	1.9	88	96	889	Y	D
190	ROJA	19	F	S	1	N	Y	N	Y	N	N	N	N	N	33	0.7	26	21	250	N	S
191	SRINIVASAN	42	M	S	1	N	Y	N	Y	N	N	N	N	N	45	0.8	35	30	136	N	S
192	AISHA	58	F	S	1	N	Y	N	Y	N	N	N	N	N	30	0.8	29	28	216	N	S
193	SIVADAS	41	M	S	3	Y	N	Y	N	Y	Y	N	N	Y	34	2.2	88	98	548	N	D
194	KUPPATHAL	38	F	S	2	N	Y	N	Y	N	N	N	Y	N	88	0.9	30	28	320	Y	S
195	SUBBAIAH	74	F	S	2	N	Y	Y	N	N	N	N	Y	N	29	1.5	26	44	227	N	S
196	SAFINA PARVEEN	56	F	S	1	N	Y	N	Y	N	N	N	N	N	42	0.9	34	32	251	N	S
197	SATHYARAJ	33	M	S	1	N	Y	N	Y	N	N	N	N	N	44	0.9	46	48	120	N	S
198	ARATHI	25	F	S	1	N	Y	N	Y	N	N	N	N	N	39	0.9	42	40	158	N	S
199	RATHNASAMY	42	M	A	1	N	Y	N	Y	N	N	N	N	N	34	1	33	32	160	N	S
200	PRIYA	36	F	S	1	N	Y	N	Y	N	N	N	N	N	33	1	35	31	194	N	S
201	SELVAM	29	M	S	1	N	Y	N	Y	N	N	N	N	N	30	0.3	38	40	120	N	S
202	BABY	53	F	S	3	Y	N	Y	N	Y	Y	N	N	Y	79	1.9	89	98	480	Y	D
203	ABUTHAHIR	49	M	S	2	N	Y	Y	N	Y	Y	N	N	N	33	0.8	48	45	201	N	S
204	MARIYAM SURAY	53	F	S	2	N	Y	Y	N	Y	Y	N	Y	N	39	1.3	52	56	324	N	S
205	SURYA	22	F	S	2	N	Y	Y	N	Y	Y	N	Y	N	29	0.9	25	22	223	Y	S
206	RAVI	47	M	S	1	N	Y	N	Y	N	N	N	N	N	32	0.7	22	20	145	N	S
207	MARY	17	F	S	1	N	Y	N	Y	N	N	N	N	N	33	0.9	26	42	120	N	S
208	SENTHIL KUMAR	25	M	A	1	N	Y	N	Y	N	N	N	N	N	30	0.9	30	25	132	N	S
209	MAHESWARI	52	F	A	3	Y	N	Y	N	Y	Y	N	N	N	66	2.6	21	100	456	Y	D
210	FATHIMA	23	F	S	1	N	Y	N	Y	N	N	N	N	N	34	0.8	32	26	195	N	S

Sl.No	NAME	AGE	SEX	MODE	APACHE 2	BP <100/60	BP 120/80	GCS<15/15	GCS15/15	SEIZURES	HYPOXIA	ECG SINUS TACHY	ST DEPRESSION	ARRYTHMIA	UREA	CREATININE	SGOT	SGPT	LDH	VENTILATION	OUTCOME
211	UMASHANKAR	44	F	S	2	N	Y	N	Y	Y	N	Y	N	N	38	1.2	36	28	218	N	S
212	LIYAKATHALI	31	M	S	2	N	Y	Y	N	Y	N	Y	N	N	50	1.6	46	42	278	N	S
213	PANDIYAMMAL	66	F	S	3	Y	N	Y	N	Y	Y	N	N	Y	63	1.9	76	102	486	Y	D
214	GOWRI	18	F	S	3	Y	N	Y	N	Y	Y	N	N	Y	56	1.9	88	112	546	Y	D
215	SANGEETHA	19	F	S	1	N	Y	N	Y	N	N	N	N	N	27	0.7	22	23	145	N	S
216	LATHAKUMARI	16	F	A	1	N	Y	N	Y	N	N	N	N	N	30	0.7	19	16	142	N	S
217	RAMPRASANTH	22	M	S	1	N	Y	N	Y	N	N	N	N	N	42	1	29	36	196	N	S
218	RAGI	15	M	H	1	N	Y	N	Y	N	N	Y	N	N	29	0.9	29	26	226	N	S
219	CHELLAMMAL	25	F	S	2	N	Y	N	Y	N	N	Y	N	N	32	0.8	24	23	156	Y	S
220	JAYASREE	36	F	S	1	N	Y	N	Y	N	N	N	N	N	39	1.4	38	22	186	N	S
221	DEVARAJ	55	M	S	3	Y	N	Y	N	Y	Y	N	N	Y	56	1.5	88	88	488	Y	D
222	SUBASHREE	39	F	S	2	N	Y	Y	N	N	Y	N	Y	N	32	0.8	39	44	235	Y	S
223	DEEPAK	41	M	S	2	N	Y	Y	N	N	Y	N	Y	N	33	0.7	22	49	163	Y	S
224	RAJI	35	F	S	1	N	Y	N	Y	N	N	N	N	N	33	0.9	51	26	156	N	S
225	SUBBU	36	M	S	2	N	Y	N	Y	N	N	N	N	N	26	0.8	45	40	355	Y	S
226	THANGAMAN	32	F	S	1	N	Y	N	Y	N	N	N	N	N	35	1	25	23	201	N	S
227	MOHANA	22	F	A	2	N	Y	N	Y	N	N	N	Y	N	25	0.9	44	40	299	Y	S
228	SAVITHA	28	F	S	1	N	Y	N	Y	N	N	N	N	N	45	1.9	39	20	201	N	S
229	VIJAYAKUMA	69	M	S	1	N	Y	N	Y	N	N	N	N	N	33	1	36	28	232	N	S
230	DEVIKA	21	F	S	1	N	Y	N	Y	N	N	N	N	N	31	0.8	28	27	220	N	S
231	SHAMIKAPOO	71	M	S	1	N	Y	N	Y	N	N	N	N	N	24	0.7	24	28	125	N	S
232	NISHANTHINI	53	F	S	1	N	Y	N	Y	N	N	N	N	N	28	0.7	20	17	185	N	S
233	BHUVANA	33	F	S	1	N	Y	N	Y	N	N	N	N	N	31	0.6	25	23	113	N	S
234	RAMACHANDR	39	M	S	1	N	Y	N	Y	N	N	Y	N	N	35	0.8	36	28	204	N	S
235	THULASI	19	F	H	1	N	Y	N	Y	N	N	Y	N	N	30	0.7	18	20	137	N	S
236	KANJAMANI	49	M	A	1	N	Y	N	Y	N	N	Y	N	N	38	1	32	29	226	N	S
237	MANJULA	36	F	S	1	N	Y	N	Y	N	N	N	N	N	31	0.9	24	19	165	N	S
238	KIRUBA	35	F	S	1	N	Y	N	Y	N	N	N	N	N	31	1	32	30	224	N	S
239	BEER MOHAM	52	M	S	1	N	Y	N	Y	N	N	N	N	N	44	1.5	42	39	294	N	S
240	DEVI	26	F	S	2	Y	N	Y	N	N	Y	Y	N	N	38	1	31	46	320	Y	S
241	SRUTHI	31	F	S	1	N	Y	N	Y	N	N	N	N	N	24	0.8	23	19	186	N	S
242	ROBERT	33	M	S	1	N	Y	N	Y	N	N	N	N	N	30	0.9	53	41	321	N	S
243	SRUTHI	34	F	S	1	N	Y	N	Y	N	N	N	N	N	33	0.9	34	32	208	N	S
244	MIZAD	32	M	S	1	N	Y	N	Y	N	N	N	N	N	30	1	21	18	143	N	S
245	KULANTHIYAMMAL	31	F	S	3	Y	N	Y	N	Y	Y	N	Y	Y	55	1.9	88	96	889	Y	D

Sl.No	NAME	AGE	SEX	MODE	APACHE 2	BP <100/60	BP 120/80	GCS<15/15	GCS15/15	SEIZURES	HYPOXIA	ECG SINUS TACHY	ST DEPRESSION	ARRYTHMIA	UREA	CREATININE	SGOT	SGPT	LDH	VENTILATION	OUTCOME
246	CHELLAM	35	F	S	1	N	Y	N	Y	N	N	N	N	N	27	0.7	22	19	164	N	S
247	SAM FRANKLIN	47	M	S	2	Y	N	Y	N	N	Y	N	Y	N	30	0.8	39	36	320	N	S
248	MAYILATHAL	51	F	S	3	Y	N	Y	N	Y	Y	N	Y	Y	89	1.1	84	90	448	Y	D
249	SARADHA	34	F	S	1	N	Y	N	Y	N	N	N	N	N	26	0.8	46	42	210	N	S
250	VINITHA	26	F	S	1	N	Y	N	Y	N	N	N	N	N	22	0.8	17	16	200	N	S
251	KALIMUTHU	32	M	S	1	N	Y	N	Y	N	N	Y	N	N	34	0.7	21	18	194	N	S
252	MEHARUNNISA	39	F	S	1	N	Y	N	Y	N	N	N	N	N	30	0.9	39	36	230	N	S
253	KRISHNA	28	F	S	3	Y	N	Y	N	Y	Y	N	N	Y	56	1.6	78	96	652	Y	D
254	BALASENTHIL	44	M	S	1	N	Y	N	Y	N	N	N	N	N	29	0.9	46	44	210	N	S
255	SANKARMANI	68	F	S	1	N	Y	N	Y	N	N	N	N	N	30	0.6	16	17	123	N	S
256	SUSIL	33	F	S	1	N	Y	N	Y	N	N	N	N	N	22	0.8	20	18	178	N	S
257	MANJULA	39	F	S	2	Y	N	N	Y	Y	Y	N	N	N	31	1	44	38	265	Y	S
258	KALAIYARASAN	29	M	S	3	Y	N	Y	N	Y	Y	N	Y	Y	88	1.9	75	98	654	Y	D
259	PAVITHRA	55	F	S	2	Y	N	Y	N	N	Y	N	N	N	45	1.2	48	39	321	N	S
260	PAVIYTHRAN	22	F	A	1	N	Y	N	Y	N	N	N	N	N	28	0.8	25	23	187	N	S
261	PREETHI	34	F	S	1	N	Y	N	Y	N	N	N	Y	N	29	0.9	36	35	245	N	S
262	SATHYA	33	F	S	2	N	Y	N	Y	N	N	N	Y	N	26	0.7	22	20	252	N	S
263	RANJITH	35	M	S	2	N	Y	N	Y	N	N	Y	N	N	33	1.2	19	39	221	N	S
264	NAVAKODEESW	46	M	S	1	N	Y	N	Y	N	N	Y	N	N	29	0.9	30	28	215	N	S
265	ANANTHAKUMA	36	F	S	3	Y	N	Y	N	Y	Y	Y	N	Y	66	1.8	78	100	662	Y	S
266	MARUTHAMMAL	69	F	S	2	Y	N	Y	N	Y	Y	N	Y	N	37	1	35	34	365	N	D
267	BHAGYALAKSH	56	F	S	2	Y	N	Y	N	Y	Y	N	Y	N	43	1.3	38	37	287	N	S
268	BALAMURUGAN	63	M	S	1	N	Y	N	Y	N	N	N	N	N	28	0.8	23	29	294	N	S
269	SIVASANKAR	47	M	S	1	N	Y	N	Y	N	N	N	N	N	30	0.8	22	26	204	N	S
270	KUPPAMMAL	58	F	S	3	Y	N	Y	N	Y	Y	N	N	Y	65	1.5	98	62	662	Y	D
271	GEORGE	49	M	S	1	N	Y	N	Y	N	N	Y	N	N	36	0.8	26	42	272	N	S
272	JAYASREE	36	F	S	1	N	Y	N	Y	N	N	N	N	N	39	1.4	38	22	186	N	S
273	DEVARAJ	55	M	S	3	Y	N	Y	N	Y	Y	N	N	Y	56	1.5	88	88	488	Y	D
274	SUBASHREE	39	F	S	2	N	Y	Y	N	N	Y	N	Y	N	32	0.8	39	44	235	Y	S
275	DEEPAK	41	M	S	2	N	Y	Y	N	N	Y	N	Y	N	33	0.7	22	49	163	Y	S
276	RAJI	35	F	S	1	N	Y	N	Y	N	N	N	N	N	33	0.9	51	26	156	N	S
277	SUBBU	36	M	S	2	N	Y	N	Y	N	N	N	N	N	26	0.8	45	40	355	Y	S
278	THANGAMAN	32	F	S	1	N	Y	N	Y	N	N	N	N	N	35	1	25	23	201	N	S
279	MOHANA	22	F	A	2	N	Y	N	Y	N	N	N	Y	N	25	0.9	44	40	299	Y	S
280	SAVITHA	28	F	S	1	N	Y	N	Y	N	N	N	N	N	45	1.9	39	20	201	N	S

Sl.No	NAME	AGE	SEX	MODE	APACHE 2	BP <100/60	BP 120/80	GCS<15/15	GCS15/15	SEIZURES	HYPOXIA	ECG SINUS TACHY	ST DEPRESSION	ARRYTHMIA	UREA	CREATININE	SGOT	SGPT	LDH	VENTILATION	OUTCOME
281	VIJAYAKUMA	69	M	S	1	N	Y	N	Y	N	N	N	N	N	33	1	36	28	232	N	S
282	DEVIKA	21	F	S	1	N	Y	N	Y	N	N	N	N	N	31	0.8	28	27	220	N	S
283	SHAMIKAPOO	71	M	S	1	N	Y	N	Y	N	N	N	N	N	24	0.7	24	28	125	N	S
284	NISHANTHINI	53	F	S	1	N	Y	N	Y	N	N	N	N	N	28	0.7	20	17	185	N	S
285	BHUVANA	33	F	S	1	N	Y	N	Y	N	N	N	N	N	31	0.6	25	23	113	N	S
286	RAMACHANDR	39	M	S	1	N	Y	N	Y	N	N	Y	N	N	35	0.8	36	28	204	N	S
287	THULASI	19	F	H	1	N	Y	N	Y	N	N	Y	N	N	30	0.7	18	20	137	N	S
288	KANJAMANI	49	M	A	1	N	Y	N	Y	N	N	Y	N	N	38	1	32	29	226	N	S
289	MANJULA	36	F	S	1	N	Y	N	Y	N	N	N	N	N	31	0.9	24	19	165	N	S
290	KIRUBA	35	F	S	1	N	Y	N	Y	N	N	N	N	N	31	1	32	30	224	N	S
291	BEER MOHAM	52	M	S	1	N	Y	N	Y	N	N	N	N	N	44	1.5	42	39	294	N	S
292	DEVI	26	F	S	2	Y	N	Y	N	N	Y	Y	N	N	38	1	31	46	320	Y	S
293	SRUTHI	31	F	S	1	N	Y	N	Y	N	N	N	N	N	24	0.8	23	19	186	N	S
294	ROBERT	33	M	S	1	N	Y	N	Y	N	N	N	N	N	30	0.9	53	41	321	N	S
295	SRUTHI	34	F	S	1	N	Y	N	Y	N	N	N	N	N	33	0.9	34	32	208	N	S
296	MIZAD	32	M	S	1	N	Y	N	Y	N	N	N	N	N	30	1	21	18	143	N	S
297	KULANTHIYAMMAL	31	F	S	3	Y	N	Y	N	Y	Y	N	Y	Y	55	1.9	88	96	889	Y	D
298	ROBERT	33	M	S	1	N	Y	N	Y	N	N	N	N	N	30	0.9	53	41	321	N	S
299	SRUTHI	34	F	S	1	N	Y	N	Y	N	N	N	N	N	33	0.9	34	32	208	N	S
300	RAMESH	40	M	S	1	N	Y	N	Y	N	N	N	N	N	33	0.9	34	32	208	N	S